draft
Environmental Impact Statement

Vegetation Treatment on BLM Lands
in Thirteen Western States

Appendixes

**United States Department of the Interior
Bureau of Land Management**

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Environmental
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Statement

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on BLM Lands
in Thirteen Western States

Prepared by
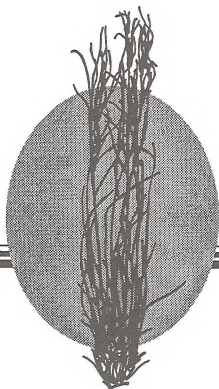**United States Department of the Interior**
Bureau of Land Management

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Appendixes

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Appendix A
Glossary

A

Absorption. The taking up of liquids by solids or the passage of a substance into the tissues of an organism as the result of diffusion, filtration, or osmosis.

Acetone. A colorless, volatile liquid that is useful as a solvent. It is found in the blood and urine when fats are improperly metabolized.

Acid Equivalent (a.e.). The amount of active ingredient expressed in terms of the parent acid.

Active Ingredient (a.i.). The chemical in an herbicide that is primarily responsible for the desired effects.

Acute Toxicity. The quality or potential of a substance to cause injury or illness shortly after exposure to a relatively large dose.

Adenoma. An abnormal growth of glandular tissue.

Adsorption. Adhesion of substances to the surfaces of solids or liquids. Technically, the attraction of ions of compounds to the surface of solids or liquids.

Adverse Impacts. Impacts that harm one or more ecosystem components or processes.

Alleopathic. Pertaining to the suppression of growth of one plant species by another through the release of toxic substances.

Alluviation. Accumulation of stream-laid sediments.

Ames Assay. A type of short-term test using bacteria in laboratory cultures to assess the mutagenic potential of a substance.

Annual Plant. A plant that completes its life cycle within 1 year.

Aquifer. A geologic formation capable of transmitting water through its pores at a rate sufficient for water supply purposes. The term water-bearing is sometimes used synonymously with aquifer when a stratum furnishes water for a specific use. Aquifers are usually saturated sands, gravel, fractures, caverns, or vesicular rock.

Archaeological and Historic Site. A site that contains either objects of antiquity or of cultural value relating to history and/or prehistory that warrant special attention.

Area of Critical Environmental Concern (ACEC). An area within public lands that requires special management attention to protect and prevent irreparable damage to important historic, cultural, or scenic values; fish and wildlife resources; other natural systems or processes; or to protect life or provide safety from natural hazards.

Arid. A term applied to regions or climates where lack of sufficient moisture severely limits growth and production of vegetation. The limits of precipitation vary considerably according to temperature conditions.

Assay. A test or measurement used to evaluate a characteristic of a chemical. See Bioassay.

Association. A group of species characterizing a certain climatologic or microclimatologic area.

B

Bajadas. Lower slopes of desert mountains.

Best Management Practice. A practice or combination of practices that is determined—after problem assessment, examination of alternative practices, and public participation—to be the most effective, practicable means of preventing or reducing the amount of pollution generated by nonpoint source to a level compatible with water quality goals.

Biennial Plant. A plant that completes its life cycle in 2 years.

Bioaccumulation. The process of a plant or animal selectively taking in or storing a persistent substance. Over time, a higher concentration of the substance is found in the organism than in the organism's environment.

Bioassay. A method for quantitatively determining the concentration of a substance

by its effect on a suitable animal, plant, or microorganism under controlled conditions.

Biological Control. The use of natural enemies to attack a target plant, retard growth, prevent regrowth, or prevent seed formation.

Biomass. The total of living organisms of one or more species per unit area or all of the species in a community measured in dry weight or kilocalories.

Boom (herbicide spray). A tubular metal device that conducts an herbicide mixture from a tank to a series of spray nozzles. It may be mounted beneath a helicopter or a fixed-wing aircraft or behind a tractor.

Broadcast Application. An application of a pesticide that uniformly covers an entire area.

Browse. The part of shrubs, half shrubs, woody vines, and trees available for animal consumption; to search for or consume browse.

Buffer Strip/Zone. A strip of vegetation that is left or managed to reduce the impact that a treatment or action on one area might have on another area.

Butte. A hill that rises above the surrounding area, with steep sloping sides and a flat top.

C

Cancer Potency. A measure of the relative ability of a substance to cause cancer. Usually expressed as a function of unit dose as per mg/kg/day. When multiplied by an estimated daily lifetime dose (in mg/kg/day) of an individual, it yields an estimate of the probability of that the individual will develop cancer.

Canopy. The uppermost cover of branches and leafy foliage in a forest.

Carcinogenic. Capable of producing or inciting cancer.

Carcinoma. A malignant or cancerous tumor.

Chemical Degradation. The breakdown of a chemical substance into simpler components through chemical reactions.

Chromosome. Microscopic structures within the cell that are composed of DNA and genes (hereditary determiners).

Chronic (effects or toxicity). Having poisonous or deleterious effects from prolonged exposure or repeated administration of a chemical.

Climax Species. The kind of plant that predominates in the final stage of ecological succession in a forest.

Cold Desert. An arid region with snow and hard frost in the winter.

Colluvium. A loose deposit of rock debris accumulated at the base of a slope.

Community. An assemblage of populations of plants and/or animals in a common spatial arrangement.

Composition. The proportions (percentages) of various plant species in relation to the total on a given area. It may be expressed in terms of cover, density, or weight.

Conifer. An order of Gymnospermae, comprising a wide range of trees, mostly evergreens that bear cones and have needle-shaped or scalelike leaves; timber commercially identified as softwood.

Conjunctivitis. Inflammation of the mucous membrane that lines the inner surface of the eyelids.

Consolidated. Material that is dense, and whose particles are cemented together.

Critical Habitat. (1) Specific areas within the habitat a species occupies at the time it is listed under the Endangered Species Act that have physical or biological features (a) that are essential to the conservation of the species and (b) that may require special management considerations or protection, and (2) specific areas outside the habitat a species occupies at the time it is listed that the Secretary of the Interior determines are essential for the species conservation.

Crossing Over. The breaking and exchanging of parts of chromosomes between chromosome pairs during cell division.

Cultural Resources. Remains of human activity, occupation, or endeavor, reflected in districts, sites, structures, buildings, objects, artifacts, ruins, works of art, architecture, and natural features that were important in past human events. Cultural resources consist of (1) physical remains, (2) areas where significant human events occurred, even though evidence of the events no longer

remains, and (3) the environment immediately surrounding the actual resource.

Cytogenetic. Refers to the structure or function of chromosomes within cells.

D

Degradation. See chemical degradation.

Demyelination/demyelinization. The destruction or removal of the myelin sheath of nerve tissue. The myelin sheath is composed of layers of myelin, a lipid material that provides electrical insulation and protection for neurons.

Dermal Exposure. That part of an amount of toxic substance that an organism receives as a result of the substance coming into contact with the organism's body surface.

Dermatitis. Inflammation of the skin.

Disclimax. An ecological community that is normally stable, which has been altered by man or other influences.

Dislodgeable Residue. A pesticide residue that can be removed from surfaces such as foliage by physical contact.

Diurnal. Pertaining to those organisms that are active during the daytime.

DNA. Deoxyribonucleic acid. Any of various nucleic acids that are the molecular basis of heredity in many organisms.

Dominant Lethal Assay. A toxicity test whereby a male animal (usually a rodent) is exposed to a chemical substance and later is sequentially mated with two female animals. The females are sacrificed, and the number and status of the fetuses is recorded.

Dose. The amount of chemical administered or received by an organism, generally at a given point in time.

Drift. That part of a sprayed chemical that is moved by wind off a target site.

Drip Torch. A container of slash-burning fuel equipped with a wick to ignite the fuel mixture as it drips from the container onto the slash. Hand-held torches have a 1.5-gallon capacity and are ignited by a fiber-filled, fuel-soaked wick.

E

Ecosystem. An interacting system of organisms considered together with their environment; for example, marsh, watershed, and lake ecosystems.

Ecotone. A transition line or strip of vegetation between two communities having characteristics of both kinds of neighboring vegetation as well as characteristics of its own.

Edaphic. Of or pertaining to soil.

Edge Habitat. The more or less well-defined boundary between two or more elements of the environment; for example, field/woodland.

Embryotoxic. Causing adverse effects at the early stages of an organism's development (embryonic period).

Endangered Species. Plant or animal species that are in danger of extinction throughout all or a significant part of their range.

Environmental Analysis. An evaluative process by which alternatives for achieving a purpose are analyzed to determine their environmental impacts.

Environmental Assessment (EA). A systematic environmental analysis of a site-specific BLM activity used to determine whether the activity would have a significant effect on the quality of the environment and whether an environmental impact statement is required.

Environmental Fate. The transport, accumulation, and disappearance of an herbicide in the environment.

Environmental Impact Statement (EIS). An analysis that assesses the probable effects of proposed actions and alternatives on the environment, in accordance with the National Environmental Policy Act.

Ephemeral Stream. A stream that flows only in direct response to precipitation and whose channel is at all times above the water table.

Ephemerals. Annual plants that complete their life cycle in a very few weeks.

Eradication. Removal of all traces of a population or elimination of a population to the point where individuals are no longer detectable.

Erosional Rills. An accelerated form of erosion with closely spaced channels scored into exposed soil.

Escarpment. A steep slope resulting from erosion or faulting that separates two level areas of different elevations.

Escherichia coli or E. coli. A common species of bacteria used in many areas of biological research, including mutagenicity testing.

Ester. A compound formed by the reaction of an acid and an alcohol, generally accompanied by the elimination of water.

Evapotranspiration. Total water loss from the soil by both transpiration from plant surfaces and direct evaporation.

Exposure Analysis. The estimation of the amount of chemical that is in an organism's environment and is available for uptake into the body.

F

F_0. In genetics and reproduction studies, it pertains to the first parents' generation.

F_1. In genetics, it refers to the first generation of offspring from the F_0 generation.

Fate. The course of an applied herbicide in an ecosystem or biological system, including metabolism, microbial degradation, leaching, and photodecomposition.

Fetotoxic. Capable of producing adverse effects in a developing fetus.

Fibroblast. Any cell from which connective tissue is developed.

Forage. Browse and herbage that is available and may provide food for animals or be harvested for feeding; to search for or consume forage.

Forb. Any nongrasslike plant having little or no woody material; or a broadleaved flowering plant whose stem, above ground, does not become woody and persistent.

Formulation. A chemical mixture that includes a certain percentage of active ingredient (technical chemical) with an inert carrier.

Friable. Soil that is brittle and easily crumbled.

Fuel. Any substance or composite mixture that can ignite and burn.

Fuel Break. A wide strip with a low amount of fuel, usually grass, in a brush or wooded area to provide soil cover and serve as a line of fire defense. It may contain a firebreak in the center.

G

Gavage. Feeding by way of a tube inserted into the stomach.

Gene. The basic unit of heredity. Each gene occupies a specific place (locus) on a chromosome.

Genotoxic. Harmful to genetic material (DNA).

Germ Cell. A functional sex cell that combines with the opposite sex cell for fertilization, for example, sperm, egg.

Global 82. A computer program by Howe and Crump (1982) used to fit the multistage or one-hit models to experimental cancer data.

Ground Water. Subsurface water that is in the zone of saturation. The top surface of the ground water is the "water table." Source of water for wells, seepage, springs.

H

Habitat. The natural abode of a plant or animal, including all biotic, climatic, and edaphic factors affecting life.

Half-life. The amount of time required for half of a compound to degrade.

Half-shrub. A plant with a woody base whose annually produced stems die each year.

Hazard. The characteristic of an item or substance that renders it capable of producing injury or illness.

Hazard Analysis. The determination of whether a particular chemical is or is not causally linked to particular harmful effects.

HDT. Highest dose tested.

Hectare (ha). 10,000 square meters, or approximately 2.47 acres.

Hematocrit. The percentage by volume of red blood cells in a given volume of blood.

Hemoglobin. The iron-containing compound in red blood cells that functions to carry oxygen from the lungs to the tissues.

Hepatoma. A tumor of the liver.

Herbaceous. A plant that does not develop persistent woody tissue above the ground.

Herbicide. A chemical used to control, suppress, or kill plants, or to severely interrupt their normal growth processes.

Herbivore. A plant-eating animal.

Heritable. Capable of being passed on from parents to offspring.

Histology. The study of the microscopic structure of tissue.

Histopathologic. Referring to tissue changes characteristic of disease.

Horizon. A layer of soil or soil material approximately parallel to the land surface and differing from adjacent related layers in physical, chemical, and biological properties and characteristics.

Hot Desert. An arid region where the winters are mild.

Hydrolysis. Decomposition or alteration of a chemical substance by water.

Hyperplasia. An excessive proliferation of normal cells in the tissue of an organ.

Hypertrophy. An increase in size of an organ or structure that does not involve tumor formation.

I

Igneous Metamorphic. Metamorphic rocks that have been physically and chemically changed by intense pressure and heat.

Illuviation. Accumulation in a lower soil horizon of materials brought down from a higher horizon.

In Vitro. Pertaining to a test that is conducted outside the living body in an artificial environment, such as a test tube or petri dish.

In Vivo. Pertaining to a test that is performed within the living body of an organism.

Integrated Pest Management. The selection, integration, and implementation of treatment methods based on predicted ecologic, sociologic, and economic effects.

Intermittent Stream. A stream that flows only at certain times of the year when it receives water from springs or from some surface source, such as melting snow.

Intraperitoneal. Related to a structure or process occurring within the peritoneum, a membranous lining of the body cavity.

Intravenous. Within or into a vein.

K

Kilogram (kg). One thousand grams, or approximately 2.2 pounds.

L

Label. All printed material on or attached to a pesticide container as required by law.

Lagomorphs. Hares and rabbits.

Land Use Plan. A plan that provides management direction on future land uses.

Latency Period. The time between a stimulus and its response.

LC_{50}. A lethal concentration rate at which 50 percent of the test animals will be killed. It is usually used in testing fish or other aquatic animals.

LD_{50}. The dosage of toxicant, expressed in milligrams of toxicant per kilogram of animal body weight, required to kill 50 percent of the animals in a test population when given orally.

LDT. Lowest dose tested.

Leach. Usually refers to the movement of chemicals through soil by water; may also refer to the movement of herbicides out of leaves, stems, or roots into the air or soil.

Least Squares Estimation. A mathematical approach used to fit a straight line (or other models) so that the sum of the squares of the vertical distances of the data points from the line will be a minimum.

Litter. The upper portion of the organic layer covering the soil, consisting of unaltered dead remains of plants and animals whose original form is still visible.

Lowest Effect Level (LEL). The lowest dose tested that results in an effect in a test organism.

Linear Regression. A mathematical procedure used to draw a straight line that best fits a set of data points on a graph.

Log-Probit Model. An equation used to describe the relationship between dose and the probability of contracting cancer. This equation can be derived by assuming that humans (or animals) have various susceptibilities, but that at very low doses none has a significant risk.

Lymphocyte. A cell of the lymphatic system, or a special type of white blood cell.

Lymphoma. A general term for the growth of new tissue in the lymphatic system.

M

Malignant. Used in reference to a tumor; indicating the presence of cancer and tending to grow worse and spread within an organism.

Margin of Safety (MOS). The ratio between the no-observed-effect level (NOEL) and the estimated dose.

Meristematic. Pertaining to the meristem, which is undifferentiated plant cells that divide to form all plant cells. All plant growth after the seed stage depends on meristematic tissue.

Mesic. Having moderate rainfall or available moisture.

Metabolism. The chemical changes in living cells by which energy is provided for vital processes and new material is assimilated.

Metabolite. A product of one metabolic process that is essential to another such process in the same organism.

Mg/kg. Milligrams per kilogram.

Mg/kg/day. Milligrams per kilogram of body weight per day.

Microbial Degradation. The breakdown of a chemical substance into simpler components by bacteria or other microorganisms.

Microclimate. The climate of a specific place within an area.

Microgram (μg). One millionth of a gram.

Mitigate. To make less harsh or harmful.

Mitigation Measures. Means taken to avoid, compensate for, rectify, or reduce the potential adverse impacts of an action.

Mitotic. Pertaining to the process of cell division that results in two cells having the same number of chromosomes as the original cell.

Monitoring. The orderly collection, analysis, and interpretation of resource data to evaluate progress toward meeting management objectives.

Montane. Growing in or inhabiting mountain areas.

Multiple Use. A resource management method that seeks more than one use from a resource area.

Multistage Model. An equation used to describe the relationship between dose and the probability of contracting cancer. This equation, commonly used by EPA, assumes that several successive events must occur to produce cancer.

Mutagen. A substance that tends to increase the frequency or extent of genetic mutations (changes in hereditary material).

Mutagenic. Capable of producing genetic defects in an organism.

Mutagenicity Assay. A study to determine whether a substance causes genetic damage.

Mutation. A change in the genetic material of a cell.

N

National Ambient Air Quality Standards (NAAQS). The allowable concentrations of air pollutants in the air specified by the Federal Government in Title 40, Code of Federal Regulations, Part 50. The air quality standards are divided into primary standards (based on the air quality criteria and allowing an adequate margin of safety requisite to protect public health) and secondary standards (based on the air quality criteria and allowing an adequate margin of safety requisite to protect the public welfare). Welfare includes effects on soils, water, crops, vegetation, manufactured materials, animals, wildlife, weather, visibility, and climate; damage to and deterioration of property; hazards to

transportation; and effects on economic values and on personal comfort and well-being.

National Wild and Scenic Rivers System. A system of nationally designated rivers and their immediate environments that have outstanding scenic, recreational, geologic, fish and wildlife, historic, cultural, and other similar values and are preserved in a free-flowing condition. This system consists of three types: (1) recreation—rivers or sections of rivers readily accessible by road or railroad that may have some development along their shorelines and may have undergone some impoundment or diversion in the past; (2) scenic—rivers or sections of rivers free of impoundments, with shorelines or watersheds still largely undeveloped but accessible in places by roads; and (3) wild—rivers or sections of rivers free of impoundments and generally inaccessible except by trails, with watersheds or shorelines essentially primitive and waters unpolluted.

Necrosis. Death of a cell or group of cells as a result of injury, disease, or other pathologic state.

Neoplastic. Pertaining to new abnormal tissue formation (neoplasms).

Neuropathy. Any disease affecting neurons, the fundamental functional units of nervous tissues.

Neurotoxic. Toxic to nerves or nervous tissue.

NOEL (no-observed-effect level). The dose level at which no toxic effects are observed in a test organism.

Nontarget Vegetation. Vegetation that is nneither expected nor planned to be affected.

Noxious Weed. According to the Federal Noxious Weed Act (PL 93-629), a weed that causes disease or has other adverse effects on man or his environment and therefore is detrimental to the agriculture and commerce of the United States and to the public health.

Nucleic Acid. A group of complex molecules found in cells, composed of phosphoric acid, sugars, and nitrogen bases. Includes DNA and RNA.

O

ODT. Only dose tested.

Omnivorous. Eating both animal and vegetable.

Omphalocele. A congenital hernia of the navel.

Oncogenic. Capable of producing or inducing tumors in animals, either benign (noncancerous) or malignant (cancerous).

Oncology. The branch of medicine for the study of tumors.

One-hit Model. An equation used to describe the relationship between dose and the probability of contracting cancer. This equation, used at one time by EPA, predicts the greatest cancer probability at low doses of all commonly used models.

Organic Material. An accumulation of decayed and resynthesized plant and animal residues with a high capacity for holding water and nutrients.

Orographic. Associated with the presence of mountains.

Ossification. The formation of bone.

P

Paleontology. A science dealing with life of past geological periods as known from fossils.

Papillary. Resembling or composed of small protuberances or elevations.

Parenteral. Injection of a substance into the body through any route other than the digestive tract.

Particulates. Finely divided solid or liquid particles in the air or in an emission; includes dust, smoke fumes, mist, spray, and fog.

Pathology. The study of the nature and cause of disease with respect to functional and structural changes.

Pedogenic. The process of soil formation.

Perennial Plant. A plant that completes its life cycle in more than 2 years.

Perennial Stream. A stream that flows continuously year round.

Persistence. The resistance of a pesticide to metabolism and environmental degradation.

Pesticide. As defined by FIFRA, any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest, and any substance or

mixture of substances intended for use as a plant regulator, defoliant, or desiccant.

pH. A numeric value that gives the relative acidity or alkalinity of a substance on a 0 to 14 scale with the neutral point at 7.0. Values lower than 7.0 show the presence of acids, and values greater than 7.0 show the presence of alkalis.

Pharmacokinetics. The study of rates of absorption, metabolic breakdown, and excretion of chemicals in animals.

Phenology. A branch of science dealing with relations between climate and periodic biological phenomena such as bird migration and plant flowering.

Photochemically Reactive. A property of substances or particles whose structures may be changed when solar energy is absorbed.

Photolysis (photodecomposition). The breakdown of a substance, especially a chemical compound, into simpler components by the action of radiant energy, such as sunlight.

Photosynthesis. Formation of carbohydrates in the tissues of plants exposed to light.

Phreatophytes. Plants that tap into the water table or other saturated zone with their roots.

Phytotoxic. Injurious or lethal to plants.

Pituitary Gland. A small, oval endocrine gland attached by a stalk to the base of the brain and consisting of an anterior and a posterior lobe. The gland secretes hormones that influence body growth and metabolism.

Playas. Flat land surfaces underlain by fine sediment or evaporite minerals deposited from a shallow lake on the floor of a topographic depression.

ppm (parts per million). A unit for measuring the concentration of a substance, such as a pesticide, in a carrier medium, such as food or water. For example, where the concentration is 1 ppm, the weight of the substance is 1 millionth the weight of the carrier medium; thus, 1 ppm is equal to 1 milligram of substance per kilogram of food or organism body weight, and it is equal to 1 milligram of substance per liter of water.

Prescribed Burning. The planned application of fire to wildland fuels in their natural or modified state, under specified conditions of fuels, weather, and other variables, to allow the fire to remain in a predetermined area and achieve site-specific fire and resource management objectives.

Pubescence. A covering of short or soft hairs.

Pulmonary. Concerning or involving the lungs.

Pyrolysis. Chemical breakdown caused in the process of combustion.

R

Rain Shadow. The region of diminished rainfall on the lee side of a mountain range, where the rainfall is noticeably less than on the windward side.

Raptors. Birds of prey, such as owls, hawks, or eagles.

Reentry. The return of a worker to an area that has recently been treated with a pesticide.

Regolith. Layer of mineral particles overlying bedrock.

Release. Freeing a tree or group of trees from competition by cutting or otherwise eliminating growth that is overtopping or closely surrounding it.

Relict. A remnant or fragment of a flora that remains from a former period when it was more widely distributed.

Renal Tubule. The functional unit of the kidney where urine is formed; nephron.

Residue. The quantity of an herbicide or its metabolites remaining in or on soil, water, plants, animals, or surfaces.

Resorption. Act of removal by absorption.

Resource Management Plan. A multiple-use plan that provides management direction for all Federal resources. It is often supplemented by more detailed, site-specific management plans for a particular land use activity, such as livestock grazing.

Rhizome. An underground root-like stem that produces roots and leafy shoots and provides a means for some plants to reproduce.

Rimrock. An overlying strata of resistant rock of a plateau that projects from the surrounding soil to form a vertical face.

Riparian. The banks and adjacent areas of water bodies, water courses, seeps, and springs. These waters provide soil moisture sufficiently in excess of that otherwise available locally to provide a more moist habitat than that of contiguous flood plains and uplands.

Risk. The likelihood that a given exposure to an item or substance that presents a certain hazard will produce illness or injury.

Risk Analysis. The description of the nature and often the magnitude of risk to organisms, including attendant uncertainty.

Riverine. Pertaining to a river.

Rosette. A cluster of leaves in crowded circles or spirals arising basally from a crown or apically from an axis with greatly shortened internodes.

Runoff. That part of precipitation, as well as any other flow contributions, that appears in surface streams, either perennial or intermittent.

Rural. A city with less than 2,500 inhabitants.

S

Safety Factor. A factor conventionally used to extrapolate human tolerances for chemical agents from no-observed-effect levels in animal test data.

Salmonella. A genus of bacteria used in mutagenicity testing.

Savanna. A grassland with scattered trees, whether as individuals or clumps; often a transitional type between true grassland and forest.

Sclerophyllus. Hard-leaved; referring to plants with evergreen leaves that are heavily cutinized or waxy and show resistance to desiccation under conditions of extreme drought.

Scoping. The process by which significant issues relating to a proposal are identified for environmental analysis. Scoping includes eliciting public comment on the proposal, evaluating concerns, and developing alternatives for consideration.

Sediment. Organic matter or soil that settles to the bottom of a liquid.

Sedimentary. Rocks that are formed from sediment or from transported fragments deposited in water.

Semiconsolidated. Material that is somewhat loosely aggregated.

Sensitive Species (Plants). Plant species not officially listed as threatened or endangered but that are undergoing a status review or are proposed for listing by either *Federal Register* notices published by the Secretary of the Interior or Secretary of Commerce or by comparable State documents.

Seral Community. One of a series of biotic communities that follow one another in time on any given area.

Shrub. A plant with persistent woody stems and a relatively low growth habit; usually produces several basal shoots instead of a single bole. A shrub differs from a tree by its low stature (less than 16 feet) and nonarborescent form.

Silviculture. The care, harvest, and regeneration of stands of timber, including preparing sites for reforestation, planting trees, controlling competing vegetation, thinning, fertilizing, controlling insects and disease, and applying various harvest systems.

Sister Chromatid Exchange (SCE). A short-term test conducted with laboratory cell cultures to assess the genetic damage caused by a chemical or physical influence.

Site Preparation. The removal of slash and/or competing vegetation and usually the exposure of bare mineral soil to prepare an area for regeneration.

Slash. The residue left on the ground after timber cutting and/or accumulating as a result of storm, fire, or other damage. It includes unused logs, uprooted stumps, broken stems, branches, twigs, leaves, bark, and chips.

Soil Compaction. The compression of the soil profile from surface pressure, resulting in reduced air space, lower water-holding capacity, and decreased plant root penetrability.

Soil Profile. A vertical section of soil that shows all horizons and parent material.

Sorption. The process of taking up or holding by either absorption or adsorption.

Spot Treatment. Application of an herbicide to a small selected area as opposed to broadcast application.

Stand. A group of trees or other growth occupying a specific area and sufficiently uniform in species composition, age, arrangement, and other conditions to be distinguishable from the forest, other growth, or other land cover on adjoining areas.

State Historic Preservation Officer (SHPO). The official within each State authorized by the State at the request of the Secretary of the Interior to act as liaison for implementing the National Historic Preservation Act of 1966.

Subchronic. The effects observed from doses that are of intermediate duration, usually 3 months (90 days).

Subcutaneous. Beneath the skin, or to be introduced beneath the skin.

Surfactant. A material that improves the emulsifying, dispersing, spreading, wetting, or other surface-modifying properties of liquids.

Systemic Herbicide. An herbicide that is moved within the plant. In a more restricted sense, refers to herbicides that are applied to the foliage and move downward through the living tissue to underground parts.

Systemic Toxicity. Effects produced as a result of the distribution of a poison or foreign substance from the point of exposure to a distant site within the body.

T

T₃. T_3. Triiodothyronine. A chemical measured in tests that evaluate the functioning of the thyroid gland.

T₄. T_4. Tetraiodothyronine. A chemical measured in tests that evaluate the functioning of the thyroid gland.

Tableland. A flat, elevated region such as a mesa or a plateau.

Target Species. Plant species of competing vegetation that is controlled in favor of desired species.

Teratogen. A substance tending to cause developmental malformations in unborn human or animal offspring.

Teratogenesis. The development of abnormal structures in an embryo.

Teratogenic. Capable of producing or inciting the development of malformations in an embryo.

Teratology. The study of malformations in organisms.

Thiourea. A colorless crystalline form of urea containing sulfur in place of oxygen.

Threatened Species. Plant or animal species that are not in danger of extinction but are likely to become so within the foreseeable future throughout all or a significant portion of their range.

Threshold. A dose or exposure below which there is no apparent or measurable adverse effect.

Threshold Limit Value (TLV). The concentration of an airborne constituent to which workers may be exposed repeatedly, day by day, without adverse effect.

Thymus. A relatively small organ located in the upper chest that is important in the development of the immune system in newborn and young animals.

Thyroid Gland. A large, ductless gland lying in front of and on either side of the trachea that secretes thyroxine, which regulates the growth of the body.

Thyroid Stimulating Hormone (TSH). A chemical secreted by the pituitary gland intended to cause the thyroid gland to produce its hormones.

Tiering. The coverage of general matters in broad environmental impact statements (such as national program or policy statements) with subsequent narrower statements or environmental analysis (such as regional program statements or, ultimately, site-specific statements). These narrower statements reference the general discussions and concentrate solely on the issues specific to the region or site.

Toxicity. A characteristic of a substance that makes it poisonous.

Toxicology. The science dealing with the study of the adverse biological effects of chemicals.

Tree. A woody perennial, usually a single-stemmed plant, that has a definite crown shape and characteristically reaches a mature

height of at least 16 feet. Some plants may grow as either trees or shrubs.

Tumor. A new growth of tissue that forms an abnormal mass and performs no physiologic function. It usually develops independent of and unrestrained by the normal principles of biological growth.

Tumorigenesis. The formation and/or development of a tumor (oncogenesis).

U

Unconsolidated. Material that is loosely arranged and whose particles are not cemented together.

Urban. A city with 2,500 or more inhabitants.

V

Volatility. The quality of evaporating readily at normal temperatures and pressures.

Volatilization. The vaporizing or evaporating of a chemical substance.

W

Wettable Powder (WP). A finely divided dry formulation that can be readily suspended in water.

Westerlies. Wind from the west.

Wilderness. An area designated by Congress as part of the National Wilderness Preservation System. Wilderness areas are generally undeveloped Federal lands that retain their primeval character and influence without improvements or human habitation.

Wilderness Study Area (WSA). A roadless area that has been found to have wilderness characteristics and that is being subjected to planning and public review to determine wilderness suitability.

X

Xeric. Having limited rainfall or available moisture.

Xerophytic. Adapted to a dry environment.

Appendix B
Scoping Summary

Summary of Scoping Comments

On July 17, 1988, a Federal Register Notice of Intent was published informing the public that the Bureau of Land Management (BLM) had formed a team to prepare an environmental impact statement on the treatment of vegetation on BLM-administered land in 13 Western States: Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, North Dakota, Oklahoma, eastern Oregon, South Dakota, Utah, Washington, and Wyoming. Shortly thereafter, each BLM State office responsible for administering land in these States distributed news releases about the EIS to appropriate local newspapers.

All 13 States issued public notices of the scoping period, during which the public submitted comments to BLM State offices. The scoping period ended August 19, 1988, for nine States: Arizona, Idaho, Nevada, New Mexico, Oklahoma, eastern Oregon, Utah, Washington, and Wyoming; and September 30, 1988, for Colorado, Montana, North Dakota, and South Dakota. At the end of the scoping period, public scoping meetings were held in Idaho, New Mexico, Oregon, Utah, and Wyoming.

Public response to a vegetation treatment program was limited. Individuals who submitted written comments urged BLM to expand the scope of the EIS to include all viable alternatives for treatment. Most of the public favor treatment of some kind to reduce or eliminate noxious weeds and/or target species of various types, but they differ on the method for accomplishing this. The ranching industry wants livestock to be considered in the EIS as a legitimate management tool in vegetation treatment. However, groups concerned with wildlife and the environment hope that the EIS will lead to some livestock reduction on public lands as a result of an alternative treatment method.

Written Comments

The following is a summary of the written comments that each State submitted.

Colorado

Colorado submitted two written comments. The State of Colorado Division of Wildlife recommended incorporating wildlife management goals and nonchemical treatment into the treatment program, and the Sheep Mountain Alliance recommended employing the principles of Integrated Pest Management and restricting chemical use.

Idaho

The Committee for Idaho's High Desert submitted the one written comment requesting that the EIS consider the loss of native vegetation species in the High Desert.

Montana

Montana submitted five written comments. The Montana Audubon Council supports vegetation treatment that would improve forage and watershed conditions, and wildlife forage, and also is interested in biological, chemical, and mechanical methods to control weeds and stimulate growth of desirable plants.

Other written comments included a letter from an individual attacking the ranching industry; another asking BLM to increase forage for ranching and protect clean water; and, finally, a general comment on leafy spurge.

New Mexico

Eight of the forms New Mexico distributed requesting comment were returned. The returned forms included the following comments: Wildlife should be protected; only burning and biological treatment should be used to enhance forage for livestock; brush control is a good alternative; herbicides and aerial application are acceptable alternatives; herbicides are favored for creosote bush; and the Integrated Management approach received support.

Nevada

The State Director offered the one written comment requesting that the scope of the EIS be expanded to include methods other than chemical treatment.

North Dakota

The one written comment received was an editorial comment against the ranching industry and was unrelated to the EIS.

Oregon

The one written submission was from the State Director who requested that the EIS be expanded to include vegetation management associated with linear and aerial rights-of-way, administrative sites, agricultural and other leases, and nonexclusive easements or agreements for other purposes.

Utah

Utah submitted four written comments. One comment supported brush control; the second supported railing trees and brush and using tebuthiuron to control sagebrush; the third supported chaining, hand cutting, and using herbicides on juniper; and the fourth supported using herbicides to control noxious weeds and allowing permittees more responsibility in determining the proper vegetation treatment.

Wyoming

The State of Wyoming offered one package of comments from several department heads. The package included comments from the following: the Geological Survey, which asked that selenium-accumulator vegetation be listed among the undesirable plants; the Governor's Range Analyst, who offered assistance; the Department of Environmental Quality, which expressed concern about water quality; and the Wyoming State Archives, Museums & Historical Department, which was interested in cultural resources.

Other comments included the following: the Wyoming Wildlife Federation suggested reducing livestock on public lands; they also were concerned about chemical and mechanical treatments and controlled burning. The Wyoming Public Lands Council and the Wyoming Wool Growers Association asked that livestock be considered a management tool; and a former grazing advisory board member asked that the EIS scope be expanded to include herbicides.

Appendix C
Nonchemical Vegetation
Treatment Methods

This appendix describes the nonchemical treatment methods that would be available for use in the Bureau of Land Management's proposed vegetation treatment program. These methods include manual, mechanical, and biological treatments, and prescribed burning. (Vegetation treatment with herbicides is described in Section E2 of Appendix E, the herbicide risk assessment.) Most areas would receive only one type of treatment, but a combination of these methods can be used, based on resource needs and legal requirements. For example, herbicide may be applied to areas to desiccate vegetation that is then burned, sprouting vegetation following burning may be sprayed with herbicide, or herbicides may be applied along with some type of biological treatment.

Manual Methods

Hand-operated power tools and hand tools would be used in manual vegetation treatment to cut, clear, or prune herbaceous and woody target species or to enhance site conditions for desired plants. Under the proposed action, approximately 5 percent of the treatment areas (about 17,000 acres) would be treated in this manner. In manual treatment, workers would cut plants above ground level; pull, grub, or dig out plant root systems to prevent subsequent sprouting and regrowth; scalp at ground level to remove competing plants around desirable vegetation; or place mulch around desirable vegetation to limit the growth of competing vegetation.

Workers in manual treatments would use tools such as the hand saw, axe, shovel, rake, machete, grubbing hoe, mattock (combination of axe and grubbing hoe), brush hook, or hand clippers. Axes, shovels, grubbing hoes, and mattocks would be used to dig up and cut below the surface to remove the main root of plants such as pricklypear or mesquite that have roots that can quickly resprout in response to surface cutting or clearing. Workers in manual treatments may also use power tools, such as chain saws and power brush saws, particularly where undesirable plants have thick stems.

Although manual treatment of vegetation is labor intensive and costly compared to prescribed burning or herbicide application, it can be far more species selective and can be used in sensitive habitats where burning or herbicides would not be appropriate or in areas that are inaccessible to ground vehicles.

Mechanical Methods

BLM would use tractors, crawler-type tractors, or specially designed vehicles with attached implements for mechanical vegetation treatments. Mechanical methods would be used on about 15 percent (56,000 acres) of the proposed vegetation treatment areas. The best mechanical method for treating undesirable plants in a particular location would depend on (1) characteristics of the undesirable species present (for example, density, stem size, brittleness, and sprouting ability); (2) need for seedbed preparation and revegetation; (3) topography and terrain; (4) soil characteristics (for example, type, depth, amount and size of rocks, erosiveness, and susceptibility to compaction); (5) climatic conditions; and (6) potential cost of improvement compared to expected productivity.

Chaining

During chaining operations, workers would use heavy (40 to 90 pounds per link) anchor chains pulled behind two crawler-type tractors in a "U" or "J" pattern. The chain may be of various sizes (generally 250 to 300 feet long) and may weigh up to 32,000 pounds. The width of each swath would vary from 75 feet to 120 feet, depending on the specifications of a given project. Chaining is best suited for crushing brittle brush and uprooting more mature, woody varieties, such as big sagebrush, mesquite, pinyon, and juniper. Chaining is versatile because it can be conducted on irregular, moderately rocky terrain with slopes of up to 20 percent. Though the uprooting and dragging of plant debris may cause moderate soil disturbance, the plant debris may be left in place to minimize runoff and erosion. Depending upon the specific site characteristics and vegetation treatment plan, the chained plant debris could also be burned to enhance the efficiency of the vegetation treatment and to facilitate vegetation seeding. Burning vegetation after

chaining could also improve scenic values and eliminate potential habitats for rodents during seeding establishment. A seed mix would be applied during most chaining projects to increase ground cover and forage production.

The standard anchor chain is best suited for mature stands of pinyon-juniper and tall stands of sagebrush where understory plants are well established. This chain can also be used with good results on other sites where a lower degree of plant removal is desired (suppression or thinning). Single chaining (one pass of the chain) would be used when desired removal of the target species is less than 50 percent. However, if the area to be treated requires a higher degree of plant removal, double chaining (two passes in opposite directions) would be used. Depending upon site conditions, the acreage chained per day would vary from 50 to 100 acres.

Modified anchor chains (often referred to as the "Ely Chain") would be used where a higher degree of plant removal is desired. This type of chain is composed of a regular ship anchor chain modified by welding 2- to 3-foot segments of railroad rail or automobile axles perpendicular to each link. It is also effective where soil scarification and seedbed preparation is necessary.

Similar to chaining, disk chaining uses a modified anchor chain with plow disks welded to alternate links. The chain is towed behind crawler-type tractors and plows a swath of about 30 feet. It would often be used in combination with cyclone seeders and roller compactors to aid in the revegetation process. The disk chain has been adapted for use on terrain that precludes use of the brushland plow (described below), especially in irregular areas and rocky soils, so it is not quite as effective in plant removal and typically achieves 75 percent vegetation suppression.

Tilling and Drilling Seed

Tilling (disk or chisel plowing) would involve the use of angled disks or pointed metal-toothed implements to uproot, chop, and mulch nearly all herbaceous vegetation. This nonselective technique would be used when complete plant removal or thinning is desired in conjunction with a seeding operation. Chisel plowing would also be used to break up soil hardpan or hard soil clay layers. Tilling has the advantage of leaving considerable mulch at or near the soil surface, which encourages the growth of newly planted seeds.

Tilling is most commonly done with a brushland plow consisting of an arrangement of angled disks on a single axle that covers a swath of about 10 feet. A crawler-type tractor or a large four-wheel drive farm tractor would be used to tow the offset disk plow, which has multiple rows of disks set at different angles to one another (USDA 1980). This treatment would normally be used on smooth terrain with deeper soils that are generally free of rocks. Sagebrush and similar shrubs are the typical target species. The brushland plow would be used where near-95-percent plant suppression is desired.

Drilling seed is a seed-planting operation that is often conducted in conjunction with tilling. The seed drills are tractor-towed or tractor-mounted implements that consist of a series of furrow openers, seed metering devices, seed hoppers, and seed covering devices. These devices on the seed drill facilitate the opening of a small furrow in the seedbed, the depositing of a measured portion of seed in the furrow, and the closing of the furrow to cover the seed. Seed drills are best suited for seed operations on smooth, well-prepared seedbeds. Areas of rocky ground, rough terrain, and brush snags limit the use of seed drills, although slower operating speeds and careful maintenance generally allows seed drills to withstand the rigors of rangeland use.

Mowing

Mowing tools would be used to cut herbaceous and woody vegetation above the ground surface. These operations would be conducted primarily along highway rights-of-way to reduce fire hazards, improve vision, prevent snow buildup, or improve the overall appearance of these areas. Rotary mowers and straight-edged cutter bar mowers may be used for these purposes. A rotary mower can cover up to a 15-foot swath, while straight-edged cutter bar mowers have a variety of swath widths. The rotary type mower would be more commonly used because of its versatility and low maintenance costs.

Roller Chopping and Cutting

Roller chopping tools use the rolling action of heavy bladed drums to cut and crush vegetation up to 5 inches in diameter. The drums would be pulled by crawler-type tractors, farm tractors, or a special type of self-propelled vehicle designed for forested areas or range improvement projects. Cutting tools include several different special shearing blades mounted on crawler tractors. Like mowing and roller chopping, vegetation would

be cut above the ground line to provide a cleared area ready for direct seeding or planting.

Blading

In blading, a crawler-type tractor blade shears off small brush at ground level. Often topsoil is scraped and removed with the brush and piled into windrows during this operation. Blading is effective only on certain undesirable plant species and is limited to relatively level areas.

Grubbing

A crawler-type tractor with a brush rake or root rake attachment would be used for grubbing. The rake attachment is a multitoothed adaptation of the standard dozer blade with a row of curved teeth projecting forward at the base of the blade. The teeth, placed below the ground surface, would be used to uproot brush and comb the roots from the soil. The effects on perennial grasses would be severe, so grubbed areas would generally be reseeded to prevent extensive runoff and erosion.

Biological Methods

Biological methods of vegetation treatment would employ living organisms to selectively suppress, inhibit, or control herbaceous and woody vegetation. This method is considered to mimic most closely the natural processes of the ecosystem because it uses plant-eating organisms and precludes the use of mechanical devices, chemical treatments, or burning of undesirable vegetation. Biological methods would be used on approximately 16 percent (60,000 acres) of BLM's vegetation treatment program areas.

Controlled grazing by herbivorous animals, such as cattle, sheep, and goats, would be the most commonly used biological method of vegetation treatment. Particular complexes of insect species may also be introduced into an area of undesirable vegetation to selectively feed on targeted plants. Other biological methods could include the use of (1) microbial and viral agents (biological herbicides), (2) plant pathogens and nematodes, (3) genetic improvement of plant adaptability and reproduction, (4) interspecific plant competition, and (5) allelopathy (plants affecting other plants through chemical inhibitors). Insect and pathogen biological vegetation treatments have been used only for the past 5 to 8 years and are still considered to be in the experimental

phase. The remaining biological methods are also experimental.

Use of Herbivorous Animals

Biological vegetation treatment by herbivores is effective when the right combination of animals, season, and stocking rate results in heavy feeding on the undesirable or less desirable plants to the competitive advantage of the desirable plants. BLM range management plans would coordinate properly timed high-intensity, short-duration grazing to prevent seed set of undesirable target plant species and reduce resource competition on desirable plant species. The numbers of livestock used in an area would be increased to a point where target vegetation is effectively removed. The effectiveness of grazing for vegetation treatment depends on (1) the size of the area, (2) the degree of treatment required, (3) the types and amounts of woody and herbaceous species present, and (4) the feeding selectivity of the animals used.

Cattle are primarily grass eaters, although they consume some shrubs and forbs. Sheep consume numerous forbs and many shrubs and grasses, while goats tend to consume large quantities of woody browse. Using a systematic plan for animal feeding, several targeted plant species' top growth can be reduced throughout the year. In addition, a balanced program of feeding of domestic livestock can provide a suitable seasonal habitat for other wildlife. Using cattle and sheep in the spring and early summer has demonstrated a thinning of understory forbs and grasses while increasing the vegetative output of desirable shrubs for winter browsing by elk and other wildlife (Vallentine 1980).

Insects

Biological manipulation of vegetation through a complex of insects has proven effective in several specific cases. However, a specific host plant-phytophagous insect relationship is necessary to reduce the population of the target plant species to a status of little or no significance. Before introduction, the insects must be determined to be (1) highly damaging to the targeted plant species, (2) highly specific to the host target plant and harmless to desirable plants, (3) able to survive in the plant's habitat, (4) free of natural parasites, and (5) apparently subject to no new parasites in the host plant's habitat (Vallentine 1980).

In most cases, a complex of five or more insects is necessary to bring a plant species down to an economic treatment level. A

period of 15 to 20 years is generally required to build up sufficient insect populations to bring about an economic treatment level. The introduction of insect complexes may regulate the target plant directly by infesting the plant's vital parts (roots, seeds, leaves) or indirectly by creating a favorable environment for infection by pathogenic agents or by reducing the plant's competitive advantage in its present environment. The purpose of biological management is not complete removal, but rather reduction of the plant to a negligible status.

Insect-plant treatment programs that have met with some success include use of the cinnabar moth and the ragwort seed fly in suppressing the growth and spread of tansy ragwort. Several insect species are being evaluated for their suitability in the management of leafy spurge in the western United States, including three species of the European flea beetle (*Apthona flava*, *Apthona cyparissiae*, and *Apthona czwalinae*); the leafy spurge hawkmoth, *Hyles euphorbiae*; the stem- and root-boring beetle, *Oberea erthyrocephala*; and the fly *Bayeria capitigena*. The thistle head weevil, *Rhinocyllus conicus*, has proven effective for controlling the musk thistle in Montana and Utah, although this weevil may also infest local native thistle species. Use of the stem-mining beetle, *Ceutorhynchus litura*, for treatment and management of the Canada thistle has been established in Montana and South Dakota (Leininger 1988).

Pathogens

Using pathogens to treat vegetation is somewhat limited in the United States, although several programs have been quite successful. The *Sclerotinia sclerotiorum* fungi is native to the United States and has been successful in suppressing spotted knapweed and Canada thistle. The pathogen *Puccinea carduorum* rust species has been used in test programs to suppress the growth of musk thistle in the United States through increased plant stress and less resistance to cold, drought, or attack by other biological agents (Leininger 1988). Similar programs under way in Utah to suppress the growth of Dyer's woad have had some success.

Prescribed Burning

Prescribed burning involves a systematic plan of using controlled ignition of selected land areas where burning of the vegetation would be expected to accomplish planned benefits. This technique of vegetation management has several advantages over other plant treatment methods. Prescribed fire is widely applicable regardless of soil rockiness, slope steepness, or terrain irregularity, provided sufficient fuel is available to carry the fire (Vallentine 1980).

All burning would be conducted in accordance with BLM's Prescribed Fire Management Policy (BLM 1988), which requires the preparation of a prescribed burning plan before every burn. Factors evaluated in these burn plans include project objectives, fuels (quantity, type, distribution, moisture content), topography (ruggedness, elevation, slope), weather (temperature, wind, humidity), time of year, smoke dispersal, and predicted fire behavior (flame length, rate of spread) (USDA 1988). BLM would use fuel models to set prescription standards for a given area and would delay treatment until natural conditions approach the optimum prescription before beginning a burn program.

The actual burn technique and ignition method used on a particular site will depend on site conditions (treatment area size, season, weather conditions, winds, target vegetation) and available labor and equipment resources. Many different ignition tools are available for prescribed burning treatments. Hand-held tools, such as pressurized kerosene drip torches, propane torches, diesel flamethrowers, flares, and ignition grenades, may be used to begin a prescribed fire. Truck- or tractor-mounted flamethrowers may also be useful where treatment areas are large and accessible.

For areas where resources permit, helicopter-borne driptorches (helitorches) may be used to release an ignited gelled fuel mixture onto the treatment area. Another technique using helicopters involves hollow polystyrene spheres containing potassium permanganate that are injected with ethylene glycol immediately before ejection. The reaction of these two chemicals ignites the polystyrene sphere and the surface fuels. The sphere ignition technique is best used for spot-firing programs.

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Appendix D
Risks From Prescribed Burning

Introduction

This section presents information on potential risks to the health of workers and members of the public from the use of prescribed burning in BLM's vegetation treatment program. The first section discusses the risks of injury or fatality for workers and the public as a result of the fire itself. The second section estimates risks to workers and the public from inhalation exposure to vegetation combustion products. The third section estimates risks from volatilization of herbicide residues.

Risks From Fire

Risks to Workers

Prescribed burning presents various hazards to ground crews, who could possibly receive injuries ranging from minor burns to severe burns that may result in permanent tissue damage. However, standard safety procedures, protective gear, and training are integrated into every prescribed fire plan and are expected to reduce or eliminate most hazards (BLM 1985). If a burn escapes and causes a wildfire, the potential is higher for severe worker injuries, including fatalities. BLM's experience with prescribed burning and use of the most up-to-date safety equipment and practices make this risk low.

Risks to the Public

If a burn escapes and causes a wildfire, members of the public in adjacent areas may be endangered. The remoteness of most burn sites and the presence of fire crews and emergency communication equipment make the risk of injury to the public extremely low.

Risks From Smoke

This section is divided into three parts: a hazard analysis, which summarizes the toxicities of the combustion products of burning vegetation; an exposure analysis, which estimates exposures to the smoke; and a risk analysis, which estimates quantifiable risks to workers and the public.

Hazard Analysis

Substances that may be found in wood smoke include particulate matter, carbon monoxide, carbon dioxide, nitrogen oxides, aldehydes, ketones, and other substances. The proportion of each varies widely, depending on factors such as moisture content in the vegetation and the temperature of the fire.

Particulate Matter

Particulate matter is a result of incomplete fuel combustion. A range of particle sizes may be present, but most are less than 10 microns in diameter. The size is related to the ability to cause adverse health effects. Larger particles are more likely to cause eye, nose, and throat irritation, but they are prevented from reaching the lungs by the body's natural defense mechanisms of respiratory mucus and ciliary movement. Fine particulate matter, with a particle diameter of less than 2.5 microns, has a greater ability to avoid these defenses and reach the lungs, where it may be deposited. About 90 percent of the particles generated by prescribed burning are less than 2.5 microns in diameter.

Gases

The gaseous components of smoke, including carbon monoxide, carbon dioxide, nitrogen oxides, and sulfur oxides, generally decompose or diffuse into the atmosphere relatively quickly. However, some constituents, such as the aldehydes discussed later in this section, may attach to the particles formed and remain more concentrated and protected from decomposition.

Polynuclear Aromatic Hydrocarbons

Polynuclear aromatic hydrocarbons, or PAHs, are of significant toxicological concern in evaluating health effects from wood smoke. The PAHs in wood smoke include at least five carcinogenic chemicals. The known carcinogenic PAHs include benzo(a)pyrene, benzo(c)phenanthrene, perylene, benzo(g,h,i)perylene, and the benzofluoranthenes.

Benzo(a)pyrene (BaP) may have the greatest overall potential for a carcinogenic effect.

Although pure BaP has not been shown to be a significantly potent carcinogen in tests in laboratory animals, its potency is greatly increased when it is administered with carbon particles (as with wood smoke inhalation). Adsorption onto particles also may inhibit photodegradation, allowing BaP to persist longer in the atmosphere. The Carcinogen Assessment Group of EPA has estimated a cancer potency value for BaP of 0.0033 per microgram per cubic meter ($\mu g/m^3$) continuous lifetime exposure (Dost 1986).

Cancer potency values for the other carcinogenic PAHs (in risk per $\mu g/m^3$) are estimated to be 0.0033 for benzo(a)phenanthrene, 0.00033 for benzofluoranthenes, 0.00033 for perylene, and 0.00033 for benzo(g,h,i)perylene (Dost 1986).

Aldehydes and Ketones

Aldehydes and ketones are ciliary toxicants that inhibit the removal of foreign material from the respiratory tract. Aldehydes are known irritants, which may be adsorbed onto the surface of particulate matter. Formaldehyde may be carcinogenic. EPA (1986) classified formaldehyde in Group B1, meaning it is a probable human carcinogen, based on findings of carcinogenicity in animal studies and limited evidence of carcinogenicity in humans. The cancer potency value for formaldehyde is 1.3×10^{-5} ($\mu g/m^3$)$^{-1}$ (EPA 1986).

Exposure Analysis

Exposures to the carcinogenic and possibly carcinogenic PAHs in smoke from burning

vegetation were estimated using methods developed by Dost (1986). Exposure estimates for members of the public assume a smoke particulate density of 0.155 mg/m^3, which is equivalent to a visibility of 2 miles. Exposure estimates for workers assume a smoke particulate density of 5.0 mg/m^3, equivalent to a visibility of 100 meters. Dost calculated the concentrations of the carcinogens in smoke in relation to particulate matter concentration. These values appear in Table D-1.

Three exposure levels were calculated for workers and for the public, based on total lifetime exposures of 30, 90, and 200 days, with an exposure of 6 hours occurring on each day. Since most prescribed fires are in remote areas, members of the public are unlikely to be exposed to more than a few in their lifetime. Thirty days of exposure would result if three nearby areas were each treated with this method once each year for 10 years. A total lifetime exposure of 90 days may result from treatment of three local areas annually for 30 years. A lifetime exposure of 200 days estimates the worst case, in which 20 treatments occur in the vicinity of the same member of the public every year for 10 years. Worker exposures were calculated using the same methods as described for the public. However, it is considerably more likely that a worker may experience a lifetime number of exposures exceeding 30, and possibly approaching 90. According to Miller (1989), a single employee may work on 2 or 3 prescribed burns per year. The exposure levels evaluated therefore correspond to 10 years and 30 years of work in prescribed

Table D-1. Concentrations of Carcinogenic PAHs in Smoke

Chemical	Smoke Density = 0.155 mg/m³ (visibility = 2 miles)	Smoke Density = 5.0 mg/m³ (visibility = 100 meters)
	Concentration (µg/m³)	
Benzo(a)pyrene	0.046	1.5
Benzo(c)phenanthrene	0.12	3.80
Benzofluoranthenes	0.016	0.52
Perylene	0.031	1.0
Benzo(g,h,i)perylene	0.096	3.15

Note: Calculated in Dost (1986).

burning. A lifetime exposure of 200 days also represents the worst case for workers.

Risk Analysis

Risks were calculated by multiplying the atmospheric concentrations of the combustion products by the total exposure time and the cancer potency value calculated for each chemical. For example, the risk to a member of the public from benzo(a)pyrene in a total of 30 exposures is calculated as follows:

BaP concentration =
0.046 µg/m³ = 0.000046 mg/m³

Lifetime exposure =
6 hours/day x 3 days/year

x 10 years =
180 hours

180 hours x 1 day/24 hours =
7.5 24-hour
exposures

A 70-year lifetime = 70 x 365 = 25,550
days

Cancer potency of BaP =
0.0033 per mg/m³ continuous lifetime
exposure

Therefore:

Concentration x exposure level x
hazard = risk
0.000046 mg/m³ x 7.5 days/25,550 days
x 0.0033 (mg/m³)⁻¹ = 4.46 x 10⁻¹¹

The risk has no corresponding units because it represents a probability—namely, the probability of developing cancer as a result of a given exposure. In the example, the risk is 4.46×10^{-11}, which equals 4.46 chances in 100 billion that the given exposure would lead to cancer.

Risks for workers and the public from the combustion products in a prescribed fire are given in Table D-2. The total risk from exposure to the smoke is the sum of the risks of the individual chemicals, which appears at the bottom of the table. In evaluating the

Table D-2. Carcinogenic Risks to Workers and Public From Vegetation Combustion Products in a Prescribed Fire

Combustion Product	Risks to Public[a] Lifetime Exposures			Risks to Workers[b] Lifetime Exposures		
	30[c]	90[d]	200[e]	30[c]	90[d]	200[e]
Benzo(a) pyrene	4.46x10⁻¹¹	1.34x10⁻¹⁰	2.97x10⁻¹⁰	1.45x10⁻⁹	4.35x10⁻⁹	9.69x10⁻⁹
Benzo(c) phenanthrene	1.16x10⁻¹⁰	3.48x10⁻¹⁰	7.75x10⁻¹⁰	3.68x10⁻⁹	1.10x10⁻⁸	2.45x10⁻⁸
Benzo- fluoranthenes	1.55x10⁻¹²	4.65x10⁻¹²	1.03x10⁻¹¹	5.04x10⁻¹¹	1.51x10⁻¹⁰	3.36x10⁻¹⁰
Perylene	3.00x10⁻¹²	9.00x10⁻¹²	2.00x10⁻¹¹	9.69x10⁻¹¹	2.91x10⁻¹⁰	6.46x10⁻¹⁰
Benzo(g,h,i) perylene	9.30x10⁻¹²	2.79x10⁻¹¹	6.20x10⁻¹¹	3.05x10⁻¹⁰	9.15x10⁻¹⁰	2.03x10⁻⁹
Total Risk	1.74x10⁻¹⁰	5.24x10⁻¹⁰	1.16x10⁻⁹	5.58x10⁻⁹	1.67x10⁻⁸	3.72x10⁻⁸

[a]Assumes that members of the public are exposed to a smoke density of 0.155 mg/m³, which is equivalent to a visibility of 2 miles.
[b]Assumes that workers are exposed to a smoke density of 5.0 mg/m³, which is equivalent to a visibility of 100 meters.
[c]Assumes exposure for 6 hours/day, 3 days/year, for 10 years.
[d]Assumes exposure for 6 hours/day, 3 days/year, for 30 years.
[e]Assumes exposure for 6 hours/day, 20 days/year, for 10 years.

increased risk of cancer from use of prescribed fire, risks are compared to the benchmark accepted in the scientific community where an increased risk of cancer is potentially significant if it exceeds 1 x 10^{-6}, or 1 in 1 million (NRC 1977).

Risk Summary

As is evident in Table D-2, which is based on the methodology developed by Dost (1986), estimated cancer risks are not expected to exceed 1 in 1 million for any worker or member of the public, even in extreme cases, from exposure to carcinogenic PAHs in the smoke from burning vegetation.

Apart from the minimal risks of cancer, possible effects on workers from smoke exposure may include eye irritation, coughing, and shortness of breath. Smoke from prescribed fires will affect air quality. Sensitive members of the public may experience eye, throat, or lung irritation from these exposures.

Risks From Herbicides in Brown-and-Burn Operations

Vegetation may be treated with herbicides several weeks before beginning a prescribed burn, with the goal of drying the vegetation to accomplish a more efficient burn. The herbicides that may be used in this method of treatment are 2,4-D, glyphosate, hexazinone, picloram, and triclopyr.

Hazard Analysis

In this assessment of risk from volatilization of herbicide residues, the exposure levels were compared to threshold limit values (TLVs), which indicate an acceptable daily exposure level for workers to airborne chemicals over their careers.

TLVs, as determined by the American Conference of Governmental and Industrial Hygienists, were available for 2,4-D and picloram. The TLV for both is 10 mg/m^3 as a time-weighted average (NIOSH 1987). The value used in this analysis for glyphosate is 12.2 mg/m^3, based on a rat inhalation LC$_{50}$ of greater than 12,200 mg/m^3 (WSSA 1983) and a safety factor of 1,000. An acceptable exposure level of 10.0 mg/m^3, as determined by DuPont (1987), was used for hexazinone. A value of 10.0 mg/m^3 was used for triclopyr, as determined in Dow (1987).

Exposure Analysis

To estimate exposure to herbicide residues from brown-and-burn operations, the following assumptions were used:

(1) All applied herbicide falls onto vegetation that will be burned as fuel in a prescribed fire.

(2) There are 44,500 kilograms of fuel per hectare, and 40 percent (17,800 kg) of it is burned (Anderson 1982).

(3) Smoke density is 5 mg/m^3 at 100 meters (Dost 1986).

(4) 50 grams of smoke are produced for each kilogram of fuel burned (USDA 1976).

(5) All herbicide residue remaining on a treated site is released into the atmosphere at the time of burning.

Based on these assumptions, the estimated volume of smoke produced is:

50 g smoke/kg fuel x 1,000 mg/g x 1 m^3/5 mg smoke
x 17,800 kg fuel/hectare = 178,000,000 m^3/hectare

The minimum time interval between herbicide application and burning for herbicides used in brown-and-burn operations in this vegetation treatment program is 30 days. Degradation rates (k) for the herbicides on vegetation were used to estimate the fraction of applied herbicide remaining at the time of burning (Table D-3).

As an example, the concentration of 2,4-D in smoke was calculated as follows:

(1) The application rate is 8 lb/acre in a forested area.

8 lb/acre x (4.536 x 10^5 mg/lb) x (2.471 acres/hectare) = 8,966,765 mg/hectare.

(2) The degradation rate is 0.0431. The time interval is 30 days. The fraction of initial herbicide remaining is

e^{-kt} = 2.7183$^{-(0.0431 \times 30)}$ = 0.2744

(3) The residue released at the time of burning is

8,966,765 mg/hectare x 0.2744 = 2,460,000 mg/hectare.

(4) Using the estimated volume of smoke produced per hectare, the 2,4-D concentration in smoke is

(2,460,000 mg/hectare)/(178,000,000 m³/hectare) = 0.01382 mg/m³.

The concentrations of the herbicides in smoke as calculated by this method are listed in Table D-4.

Risk Analysis

To evaluate risks from the use of herbicides, the atmospheric concentration of each herbicide was compared to the corresponding TLV. The TLVs are assumed to be acceptable exposure levels. Therefore, if the ratio of the TLV to the estimated exposure is greater than one, the risk is assumed to be nonsignificant. These ratios are listed in Table D-5.

All estimated exposures are significantly less than the levels determined to be safe exposure levels. These risks were calculated using a smoke density that is likely to occur onsite and therefore represent risks to workers. Members of the public would be exposed to much lower atmospheric concentrations than these and would have a margin for safety that is even greater than that calculated for workers. Based on this method of risk estimation, neither workers nor the public are expected to be at risk from the herbicide residues volatilized in a brown-and-burn operation.

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Table D-3. Properties of Herbicides Considered for Use in Brown-and-Burn Operations

Herbicide	Application Rate[a] (lb/acre)	Degradation Rate (k)[b]	TLV (mg/m³)
2,4-D	8	0.0431[c]	10[g]
Glyphosate	3	0.0495[d]	12.2[h]
Hexazinone	3	0.0584[c]	10.0[i]
Picloram	2	0.0693[e]	10[g]
Triclopyr	4	0.0040[f]	10[j]

[a]Application rate for a forested area.
[b]k = fraction of remaining herbicide that is lost through degradation or other processes per day.
[c]USDA 1984.
[d]Newton and Dost 1981.
[e]Bovey et al. 1967.

[f]Newton et al. 1982.
[g]NIOSH 1987.
[h]WSSA 1983 (divided by a safety factor of 1,000).
[i]Acceptable exposure level (Du Pont 1987).
[j]Dow 1987.

Table D-4. Concentrations of Herbicides in Smoke From Brown-and-Burn Operations at a Distance of 100 Meters From the Fire

Herbicide (mg/m³)	Concentration
2,4-D	0.01382
Glyphosate	0.004279
Hexazinone	0.003276
Picloram	0.001575
Triclopyr	0.02234

Table D-5. Ratio of Threshold Limit Value to Exposure Level for Herbicides Used in Brown-and-Burn Operations

Herbicide	TLV/estimated exposure	Risk
2,4-D	724	Negligible
Glyphosate	2,851	Negligible
Hexazinone	3,053	Negligible
Picloram	6,349	Negligible
Triclopyr	448	Negligible

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Appendix E—Risk Assessment

Section E1
Introduction

Purpose

The purpose of this appendix is to assess the risks to human health and nontarget organisms, including fish, wildlife, and domestic animals, from the use of 19 herbicides and 2 carriers, diesel oil and kerosene, in BLM's vegetation treatment program.

The analysis of the potential human health effects of using herbicides to treat vegetation was accomplished using the methodology of risk assessment generally accepted by the scientific community. In essence, the risk assessment compares the doses people may get from applying the herbicides (worker doses) or from being near an application site (public doses) with doses shown to cause no observed adverse effects in tests on laboratory animals. Estimated doses to nontarget organisms were compared to laboratory-determined median lethal doses (LD_{50}s) or median lethal concentrations (LC_{50}s).

Organization of This Appendix

This section presents the purpose, describes the structure, and outlines the methodology of the herbicide risk assessment. Section E2 describes the vegetation treatment methods that use herbicides. Section E3, the human hazard analysis, discusses the toxic properties of each herbicide as they relate to humans, including the cancer potency of each herbicide known or suspected to produce cancer in laboratory animals. Section E4, the human exposure analysis, describes how human exposures to the program herbicides were estimated. Section E5, the human health risk analysis, describes how human health risks were estimated and discusses risks to workers and the public from herbicide exposures. Section E6, the nontarget species hazard analysis, discusses the toxic properties of each herbicide to wildlife and aquatic species. Section E7, the nontarget species exposure analysis, describes how wildlife and aquatic species exposures were estimated. Section E8, the nontarget species risk analysis, describes the methodology for assessing nontarget species risks and the estimated risk to nontarget species.

Overview of the Risk Assessment

This risk assessment examines the potential health effects to humans and nontarget organisms that might be exposed to herbicides as a result of BLM vegetation program treatments. The exposed human population at risk is divided into two groups. The first group—the public—includes passersby or nearby residents. The second group—workers—includes aerial and ground applicators, fuel truck operators, and other personnel directly involved in applying herbicides. In addition, risks to birds, mammals, amphibians, insects, fish, and aquatic invertebrates were analyzed based on a comparison of laboratory toxicity studies to estimated exposures of a group of representative species.

The risk assessment includes analyses of a range of possible exposures to herbicides—from those exposures most likely to occur to those that are extremely unlikely. Assumptions about the characteristics of typical herbicide applications ("routine-realistic") are used to estimate the doses to nearby members of the public and to workers that may occur as a result of routine operations. A second set of assumptions, based on extreme values of the routine application characteristics ("routine-worst case"), is used to estimate maximum exposures that are not likely to be exceeded except in the case of an accident. A third set of assumptions about accidents is used to estimate doses to the public and workers that may result from direct exposure to the herbicide mix or concentrate.

Health risks to humans were evaluated by comparing dose estimates for the public and workers with appropriate toxicity levels as determined in tests on laboratory animals. This analysis estimates the risk of chronic health effects arising from a single exposure or from repeated exposures over various time periods for each herbicide. In addition, toxicity data for seven of the herbicides (amitrole, atrazine, bromacil, 2,4-D, glyphosate, picloram, and simazine) and the petroleum distillates kerosene and diesel oil were analyzed to estimate the risk of carcinogenicity as a result of estimated lifetime exposures. The risk of heritable mutations was evaluated qualitatively based on the weight of evidence from available data from laboratory animal studies.

Exposures to herbicide mixtures was also examined in a qualitative discussion of synergistic effects.

Risks to wildlife and aquatic species from the herbicides were determined by comparing estimated exposures to lethal levels observed in laboratory animal and field studies.

Structure of the Risk Assessment

Assessing the risk of effects from using herbicides in the vegetation treatment program requires estimating possible types of exposures that could occur as a result of herbicide applications and associated activities and estimating the probability and extent of adverse effects as a result of those exposures. This risk assessment employs the three principal analytical elements described by the National Research Council (1983) that are necessary to characterize the potential adverse health effects of exposures to existing or introduced hazards in the environment: the hazard analysis, the exposure analysis, and the risk analysis. These elements are briefly described as follows:

(1) **Hazard Analysis** requires gathering the information used to determine the toxic properties of each herbicide. Human hazard levels are derived primarily from the results of laboratory studies of animal models, such as rats, mice, and rabbits, supplemented, where appropriate, with information on human poisoning incidents, epidemiology studies, field studies of other organisms, and data on chemical structure. Nontarget species hazard levels are drawn from laboratory studies and field studies.

(2) **Exposure Analysis** involves estimating single and multiple exposures to persons and nontarget species potentially exposed to the herbicides and determining the doses likely to result from those estimated exposures.

(3) **Risk Analysis** requires comparing the hazard information with the dose estimates to predict the health effects under the given conditions of exposure.

The relationships among these three components are illustrated in Figure E1-1. This risk assessment identifies uncertainties, such as areas where scientific studies are unavailable, and describes how those uncertainties were dealt with to produce the results of the analyses. The following

discussion briefly describes how each component in the structure was addressed in this risk assessment.

Hazard Analysis

The human hazard and nontarget species hazard analyses are presented in Sections E3 and E6, respectively. The hazard involved in using each of the herbicides was determined from reviewing the results of extensive literature searches. In addition, all relevant data submitted to EPA in support of the registration of these pesticides were reviewed. These data were reviewed for required toxicity reference levels, particularly rat and nontarget species median lethal doses or LD_{50}s (a median lethal dose is the amount of a substance that will kill 50 percent of a laboratory test population), systemic and reproductive no-observed-effect levels (NOELs), and data about cancer and mutagenicity. Where scientific uncertainty exists for a particular herbicide on a specific toxic effect (mutagenicity, for example), the area is identified and a tentative conclusion is drawn from the available data on the possible affect. Where no data exist on a certain toxic endpoint, it is assumed that the chemical might cause that effect. Cancer potency values were identified or computed directly from the laboratory animal tumor data for the herbicides that have demonstrated the potential to induce an oncogenic response in mammals.

Exposure Analysis

To assess the risks from exposures to the program herbicides, various aspects of the vegetation treatment program that employ herbicides were examined. Principal aspects of the herbicide application methods that determine potential levels of exposure were identified, including human activities in or near treated areas, application rates, the size and configuration of treated areas, and mitigation measures. A discussion of the herbicide application methods that would be used in the BLM program appears in Section E2 of this appendix.

In the exposure analysis, realistic and worst case dose estimates were made for routine application operations. Doses from accidents also were estimated. Two human populations (the public and workers) may be exposed to herbicides in BLM's proposed program. For the analysis of public health effects, dose estimates were made for people assumed to

Hazard Analysis

- Identify what kind of health effects have been observed in laboratory animals and at what levels of exposure

- Identify any health effects that have been observed in humans

- Determine median lethal dose (LD$_{50}$) for acute effects from laboratory rat study

- Determine lowest no-observed-effect levels (NOELs), if possible, for general chronic toxic effects, reproductive effects, and birth defects

- Determine whether the herbicide potentially causes cancer or mutations

- Identify data gaps in toxicity information

Exposure Analysis

- Identify people exposed

- Identify routes of exposure

- Estimate how much each person would receive by each exposure route using both typical and worst case scenarios

- Estimate frequency and duration of exposure

- Calculate doses

Risk Analysis

- Compare doses to NOELs and LD$_{50}$s and discuss probability of acute and chronic effects (including birth defects) for realistic, worst case, and accident scenarios

- Conduct worst case analysis for cancer risk

Figure E1-1. Components of the risk assessment process.

be exposed because of routine operations through one or more of the following routes:

(1) Dermal, from receiving herbicide spray drift

(2) Dermal, from touching sprayed vegetation

(3) Dietary, from consuming berries that have received herbicide drift

(4) Dietary, from drinking water with drift residues

(5) Dietary, from eating fish from a pond that has received drift

For each of the above routes, realistic and worst case exposures were calculated, varying parameters such as application rate, size of treatment area, and drift conditions. For the analysis of potential risks to workers, routine doses were estimated for the following workers:

In aerial applications:

(1) Pilots

(2) Mixer-loaders

(3) Fuel truck operators

In backpack applications:

(4) Applicators

In ground mechanical applications:

(5) Applicators

(6) Mixer-loaders

(7) Applicator/mixer-loaders

In hand applications:

(8) Applicators

Because all human activities involve the possibility of error, using herbicides in vegetation treatment operations involves the possibility that humans may inadvertently receive unusually high exposures in an accident. To examine the potential health effects that could occur in an accident, the following accidents were analyzed:

(1) Spills of pesticide concentrate and mix on a person's skin

(2) Direct spraying of a worker from a broken hose

(3) Direct spraying of a person from aerial application

(4) Immediate reentry to sprayed area

(5) Consumption of water from a pond that has been aerially sprayed, that has received an 80-gallon spill of herbicide from an aerial applicator, or that has received a spill from a tank mix truck

(6) Consumption of berries that have been directly sprayed

Doses to terrestrial wildlife are considered in the nontarget species analysis, using routes of exposure that include consumption of contaminated food items, dermal exposure from spray drift and vegetation contact, and inhalation exposure. Impacts to aquatic life also are evaluated using estimated herbicide concentrations in stock ponds that either receive drift, are directly sprayed, or are contaminated by a helicopter jettison of herbicide mix.

Risk Analysis

Human health risks of the BLM vegetation treatment program were evaluated by comparing the doses of the public and workers, calculated for routine and accidental exposure scenarios, to the laboratory-determined toxicity levels described in the hazard analysis.

Many factors contribute to the uncertainty in this process of judging risks to human health from laboratory animal studies. First, the reference levels established in the laboratory are the result of tests on laboratory animals, particularly rats and mice, in which dose levels produce no observed effects. To allow for the uncertainty in extrapolating from these NOELs in laboratory animals to levels deemed acceptable for humans, safety factors are used. The generally accepted factors (NRC 1986) are 10 for moving from animals to humans (between species variation) and another 10 to account for possible variation in human responses (within species variation). This 10 times 10, or hundredfold, safety factor means that the laboratory NOEL dose reduced one hundredfold would normally be considered an acceptable or reasonably safe human dose. In this risk assessment, a margin of safety (MOS) or "hazard level to exposure level" ratio has been calculated for each estimated dose by dividing the animal NOEL by the estimated

dose. The computed MOS is then compared to the hundredfold safety factor. MOSs above 100 are assumed to indicate low risks to human health.

The risk of a pesticide causing cancer was evaluated differently. It was assumed that an herbicide that has demonstrated the potential to induce tumors in laboratory animals has some probability of inducing them at any dosage level. Animal studies were used to determine the relationship between carcinogenic risk and exposure; the laboratory data then were adjusted to reflect the lower dose ranges, larger size, and longer lifespan of humans. The risk of cancer was calculated for various categories of people that may be exposed to the herbicides in realistic and maximum exposure scenarios, with total exposure averaged over a 70-year lifetime.

The risk of heritable mutations was evaluated qualitatively on the weight of evidence from available test data on bacteria, yeasts, mammalian cells in culture, and animals. However, the mutagenicity risk was not quantified. Where appropriate, that risk is compared with the chemical's cancer risk. Cumulative risk for individuals is discussed in terms of lifetime exposures to a given herbicide for members of the public and for workers. Risk of synergistic effects is discussed in terms of the available evidence of enhanced toxicity when any of these herbicides is mixed with another chemical.

Risks to terrestrial wildlife species were evaluated by comparing estimated doses with the LD_{50} for the most closely related laboratory-tested species. Risk evaluations for aquatic species compared the herbicide's concentration in water with the concentration known to be lethal to 50 percent of the organisms of a species tested (LC_{50}). The results of these comparisons were judged according to the criteria of 1/5 LD_{50} for terrestrial species and 1/10 LC_{50} for aquatic species set forth by EPA (1986).

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Section E2
Vegetation Treatment Programs

This section describes BLM's herbicide vegetation treatments in the 13 Western States. The first subsection briefly describes the different types of vegetation treatments that use herbicides. The second subsection identifies the application methods and principal herbicides used. The final subsection discusses mitigation measures used to minimize the possible adverse effects of the herbicides on human health and the environment.

Treatment Objectives

Herbicides are used in rangeland improvement and silvicultural practice to improve the potential for success of desired vegetation by reducing competition for light, moisture, and soil nutrients by less desirable plant species. They are used to manage or restrict noxious plant species and to suppress vegetation that interferes with manmade structures or transportation corridors.

Noxious weed management programs regulate the occurrence of those noxious and poisonous plants harmful to native vegetation, humans, or domestic livestock. Plants most often treated are poison oak, tansy-ragwort, St. Johnswort, skeleton weed, and thistle. BLM's noxious weed control program in the Northwest is analyzed in a separate EIS, *Northwest Area Noxious Weed Control Program* (BLM 1985). This EIS covers noxious weed control in the rest of the 13 States. The noxious weed and poisonous plant control program is included as part of the vegetation treatment methodology that BLM uses to maintain the areas under its jurisdiction. BLM uses herbicides as one of the tools in its noxious weed management program and uses them in varying degrees in all land treatment categories.

Rangeland vegetation treatment operations provide forage for domestic livestock and wildlife by removing undesirable competing plant species and preparing seedbeds for desirable plants. Approximately 89 percent of the herbicide-treated acreage in the proposed Bureau of Land Management vegetation treatment program falls in the range improvement category in the 13 Western States. Certain aspects of BLM's range improvement program are evaluated in a separate EIS (BLM 1985).

Public domain forest land vegetation treatment operations, designed to ensure the establishment and healthy growth of timber crop species, are one of BLM's least extensive programs for herbicide treatment (Table E2-1).

Table E2-1. Annual Acres Treated by Herbicide Application Methods in the Proposed Vegetation Treatment Program (Alternative 1)

Application Method	Project Type				
	Range Improvement	Public Domain Forests	Right-of-Way Maintenance	Oil & Gas Site Maintenance	Recreation Site Maintenance
Aerial					
Fixed-Wing	58,400	300	—	—	—
Helicopter	54,725	450	700	100	—
Ground					
Vehicle	9,125	405	7,065	4,125	325
Hand	3,480	330	935	510	540
Total	125,730	1,485	8,700	4,735	865

These operations include site preparation, plantation maintenance, conifer release, precommercial thinning, and noncommercial tree removal. Site preparation treatments prepare newly harvested or inadequately stocked areas for planting new tree crops. Herbicides used in site preparation reduce vegetation that would compete with conifers. In the brown-and-burn method of site preparation, herbicides are used to dry the vegetation, which will be burned several months later. Herbicides are used in plantations some time after planting to promote the survival and establishment of conifers (maintenance) or to promote the dominance and growth of already established conifers (release). Precommercial thinning reduces competition among conifers, thereby improving the growth rate of desirable crop trees. Noncommercial tree removal is used to eliminate dwarf mistletoe-infected host trees. These latter two silvicultural practices primarily use manual methods. Herbicide use in public domain forests constitutes less than 4 percent of the vegetation treatment operations in the proposed BLM program.

Right-of-way treatments include roadside maintenance and maintenance of power transmission lines, waterways, and railroad corridors. In roadside maintenance, vegetation is removed or retarded from ditches and shoulders to prevent brush encroachment into driving lanes, to maintain visibility on curves for the safety of vehicle operators, to permit drainage structures to function as intended, and to facilitate maintenance operations. Herbicides have been used in nearly 50 percent of BLM's roadside vegetation treatment and maintenance programs in the 13 Western States.

Oil and gas drilling site and production area vegetation treatments are done to reduce potentially flammable vegetation. Vegetation treatments in these areas may include the preparation and regular maintenance of areas for use as fire control lines or fuel breaks, or the reduction of vegetation species that could pose a hazard to fire control operations. More than 50 percent of the vegetation treatment programs for oil and gas drilling sites are herbicide applications.

Recreation and cultural site maintenance operations provide for the safe and efficient use of BLM facilities and recreation sites and for permittee/grantee use of public amenities, such as ski runs, waterways, and utility terminals. Vegetation treatments are made for the general maintenance and visual appearance of the areas and to reduce

potential threats to the site's plants and wildlife, as well as visitors' health and welfare. This site maintenance program includes the noxious weed and poisonous plant control program. Vegetation treatments in these areas are also used for fire management. BLM uses herbicides on approximately one-third of the total recreation site acreage identified as needing regular vegetation treatment operations. All of the chemically treated acreage in this category uses ground-based application methods because aerial applications are not designated for these areas.

Application Methods and Herbicide Usage

BLM conducts pretreatment surveys in accordance with BLM Handbook H-9011-1 before making a decision to use herbicides on a specific land area. The chemicals can be applied by a number of different methods, and the selected technique is dependent upon a number of variables. Some of these are: (1) the treatment objective (removal or reduction); (2) accessibility, topography, and size of the treatment area; (3) the characteristics of the target species and the desired vegetation; (4) the location of sensitive areas in the immediate vicinity (potential environmental impacts); (5) the anticipated costs and equipment limitations; and (6) the meteorological and vegetative conditions of the treatment area at the time of treatment.

Nineteen herbicides are proposed for use to treat vegetation under BLM's preferred alternative. Each of the 19 herbicides has particular characteristics, including the chemical active ingredient, the formulations to be used, and the range of application rates for rangeland, public domain forest land, oil and gas sites, rights-of-way, and recreational and cultural sites (Tables E2-2 and E2-3).

Herbicide applications are scheduled and designed so that there will be minimal potential impacts on nontarget plants and animals, while remaining consistent with the objective of the vegetation treatment program. The rates of application are dependent upon the target species, the presence and condition of nontarget vegetation, the soil type, the depth to the water table, and presence of other water sources.

Herbicides are applied either from the air or on the ground. The herbicide formulations may be in a liquid or granular form depending upon resources and program objectives. Aerial

Table E2-2. Typical Herbicide Application Rates for BLM Vegetation Treatment Programs

Herbicide	Application Rate (pounds active ingredient/acre)				
	Rangeland	Public-Domain Forest Land	Oil and Gas Sites	Rights-of-Way	Recreation and Cultural Sites
Amitrole	2	2	4	2	—
Atrazine	1	4	10	4	1
Bromacil	—	—	8	8	—
Chlorsulfuron	—	0.125	0.141	0.141	0.125
Clopyralid	0.5	—	12	12	—
2,4-D	4	4	4	4	3
Dalapon	3	4	4	4	4
Dicamba	4	4	8	4	4
Diuron	—	—	10	4	—
Glyphosate	4	2	4	4	4
Hexazinone	0.67	2	4	2	2
Imazapyr	1	1.5	1.5	1.5	1.5
Mefluidide	—	—	0.25	0.25	—
Metsulfuron methyl	—	—	0.075	0.075	—
Picloram	2	2	3	3	2
Simazine	—	4	10	4	1
Sulfometuron methyl	—	—	0.563	0.563	—
Tebuthiuron	0.5	1.5	8	1.5	0.5
Triclopyr	1.5	2	4	4	1.5
Carriers					
Diesel oil	2	2	2	2	2
Kerosene	2	2	2	2	2

Table E2-3. Maximum Herbicide Application Rates for BLM Vegetation Treatment Programs

Herbicide	Application Rate (pounds active ingredient/acre)				
	Rangeland	Public-Domain Forest Land	Oil and Gas Sites	Rights-of-Way	Recreation and Cultural Sites
Amitrole	2	2	9.9	9.9	—
Atrazine	1	4	40	40	1
Bromacil	—	—	16	16	—
Chlorsulfuron	—	0.125	0.141	0.141	0.125
Clopyralid	0.5	—	12	12	—
2,4-D	6	8	4	4	3
Dalapon	3	4	22	22	4
Dicamba	8	4	8	8	8
Diuron	—	—	32	32	—
Glyphosate	5	3	4	4	5
Hexazinone	0.67	3	10.8	10.8	3
Imazapyr	1	1.5	1.5	1.5	1.5
Mefluidide	—	—	0.25	0.25	—
Metsulfuron methyl	—	—	0.075	0.075	—
Picloram	2	2	3	3	2
Simazine	—	4	40	40	4
Sulfometuron methyl	—	—	0.563	0.563	—
Tebuthiuron	4	5	16	16	4
Triclopyr	1.5	4	8	8	1.5
Carriers					
Diesel oil	2	2	2	2	2
Kerosene	2	2	2	2	2

methods employ boom-mounted nozzles for liquids or rotary broadcasters for granular formulations, carried by helicopters or fixed-wing aircraft. Ground application methods include vehicle-mounted, backpack, and hand application techniques. Vehicle-mounted application systems use fixed-boom or hand-held spray nozzles mounted on trucks or tractors. Backpack systems use a pressurized sprayer to apply an herbicide as a broadcast spray directly to one or a group of individual plants.

The principal hand application techniques are injection and stump treatment. Injection involves applying an herbicide with a hand-held container or injector through slits cut into the stems of target plants. Individual stem treatment by the injection method also is used for thinning crop trees or removing undesirable trees. Stump treatment entails directly applying liquid herbicide to the cut stump of the target plant. An herbicide can be applied by dabbing or painting the exposed cambium of a stump or by using a squeeze bottle on a freshly cut cambium surface to inhibit sprouting. Herbicides may also be applied by hand in solid form as granules spread on the ground surface. All of the herbicide application methods would be used in every type of management operation, except aerial methods on recreation sites.

Aerial Application Methods

Historically, BLM has used aerial application methods in more than 75 percent of its herbicide treatment programs. BLM treats more than 98 percent of its range management sites by air. Helicopters are preferred on rangeland projects (more than 60 percent of the time) because the many treatment units are far apart and are often small and irregularly shaped. Contractor-operated helicopters or fixed-wing aircraft are equipped with an herbicide tank or bin (depending on whether the chemical is a liquid or granular formulation). The size and type of these aircraft may vary, but the equipment used to apply the herbicides must meet specific guidelines. For aerial spraying, the aircraft would be equipped with cylindrical jet-producing nozzles no less than 1/8-inch in diameter. The nozzles would be directed with the slipstream, at a maximum of 45 degrees downward for fixed-wing, or up to 75 degrees downward for helicopter application, depending upon the flight speed. Nozzle size and pressure would be designed to produce droplets with a diameter of 200 to 400 microns. For fixed-wing aircraft, the spray boom is typically 3/4 the wing span, and for

helicopters the spray boom is often 3/4 the rotor diameter. All spray systems must have a positive liquid shut-off device that ensures that no chemical continues to drip from the boom once the pilot has completed a swath. The nozzles are spaced to produce a uniform pattern for the length of the boom.

Using helicopters for herbicide application is often more expensive than using fixed-wing aircraft, but helicopters offer greater versatility. Helicopters are well adapted to areas dominated by irregular terrain and long, narrow, and irregularly shaped land patterns, a common characteristic of BLM-managed land. Various helicopter aircraft types could be used, including Bell, Sikorsky, and Heilier models. The helicopter must be capable of accommodating the spray equipment and the herbicide tank or bin (depending on whether the chemical is in liquid or granular form) and of maintaining an airspeed of 40 to 50 miles per hour at a height of 30 to 45 feet above the vegetation (depending upon the desired application rate), and must meet BLM safety performance standards.

Fixed-wing aircraft include the typical, small "crop-duster" type aircraft, usually a Cessna model or equivalent. Fixed-wing aircraft are best suited for smoother terrain and larger tracts of land where abrupt turning is not required. Because the fixed-wing aircraft spraying operations are used for treating larger land areas, the price per acre is generally lower than for helicopter spraying. Aircraft capability requirements for fixed-wing aircraft are similar to helicopter requirements, except that an air speed of 100 to 120 miles per hour is necessary, with spraying heights of 10 to 40 feet generally used to produce the desired application rates.

Batch trucks are an integral part of any aerial operation. They serve as mixing tanks for preparing the correct proportions of herbicide and carrier, and they move with the operation when different landing areas are required.

The number of workers involved in a typical aerial spray project varies according to the type of activity. A small operation may require up to 6 individuals, while a complex spray operation may require as many as 20 to 25 workers. An aerial operations crew for range management, noxious weed management, and right-of-way maintenance usually consists of five to eight individuals. Typically, personnel on a large project include a pilot, a mixer-loader, a contracting officer's representative (COR), an observer-inspector, a one- to six-member flagging crew, one or two

law enforcement officers, one or two water monitors, and one or two laborers. Optional personnel include an air operations officer, a radio technician, a weather monitor, and a recorder.

Ground Application Methods

BLM does not use ground herbicide application methods as extensively as other agencies, such as the Forest Service. In vegetation treatment projects, ground herbicide applications normally constitute about 25 percent of the total area that BLM treats chemically.

Backpack treatment is the predominant ground-based method used for silviculture and range management. Pressurized backpack treatment operations typically involve a supervisor (who may also function as a mixer-loader), an inspector, a monitor, and 2 to 12 crew members. Backpack sprayers can typically treat one-half acre per hour in silviculture operations.

Stump treatment and herbicide injection are commonly used in forested areas. Some of the applied herbicides may also be in granular form, broadcast by hand or mechanical spreader. Four laborers and one inspector generally make up the work force for stump treatment or injection.

Right-of-way maintenance projects frequently use vehicle-mounted application techniques. A truck with a mixing/holding tank uses a front-mounted spray boom or a hand-held pressurized nozzle to treat roadside vegetation on varying slopes. However, using this equipment for off-road right-of-way projects is limited to gentle slopes (less than 20 percent) and open terrain. Contractors spray an average of 30 to 50 acres per day with vehicle-mounted applicators. A driver/mixer-loader and applicator constitute the typical crew for truck spraying.

Most noxious weed control programs are conducted on rangelands and recreational areas. Backpacks, spray bottles, and trucks or tractors with spray booms or tractor-mounted attachments are used in ground-based noxious weed programs. Typically, backpack sprayers can treat only 1 acre every 3 to 4 hours in noxious weed control programs because target plants are found as scattered individuals or in small groups. Part of the total acres treated in noxious weed control projects is hand treated with granular herbicides.

Vehicle Treatments

Herbicide treatments may use ground-based spray application techniques. Vehicular application would be made using a boom with several spray nozzles or a hand gun with a single nozzle. Ground vehicle spray equipment can be mounted on all terrain vehicles (ATVs), farm tractors, or trucks. Because of its small size and agility, the ATV can be adapted to many different situations.

The boom spray equipment used for vehicle operations is designed to spray wide strips of land where the vegetation normally does not exceed 18 inches in height and the terrain is generally smooth and free of deep gullies. Ground spraying from vehicles would frequently occur along highway rights-of-way, oil and gas sites, public domain forests, or rangeland areas.

Ground spraying operations would also be conducted from vehicles using spot-gun spraying. The spot-gun technique is best adapted for spraying small scattered individual plots. It also may be used in spraying sign posts and delineators within highway rights-of-way, and around wooden power line poles as a means of reducing fire hazards within power line rights-of-way. This technique also would be used to treat scattered noxious weed vegetation, but it is limited to those areas that are accessible by vehicles.

Hand Treatments

Other ground spraying operations require the use of a backpack-carried spray tank for carrying the herbicide mix with a spray boom or a hand gun with a single nozzle for herbicide application. These techniques are best adapted for very small-scale spraying in isolated spots and those areas that are not accessible by vehicle. They are primarily used for spot treatments around sign posts, spraying competing trees in public domain forests, delineators, power poles, scattered noxious weeds, and other areas that require selective spraying.

Mitigation Measures

Mitigation measures are intended to ensure the proper and safe application of herbicides on BLM lands in the program States and are required by Federal, State, and regional procedures. Federal and State laws and regulations set minimum standards to follow when applying herbicides on Government-owned forests and rangelands. Each regional

and district office may develop additional restrictions and precautions. The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) requires pesticide manufacturers to register their chemicals with the Federal Government and list the allowable uses, application rates, and special restrictions on each herbicide's label. All of the herbicides considered in this risk assessment are registered with the Environmental Protection Agency; and their label rates, uses, and handling instructions must be complied with according to Federal law.

The Department of the Interior, Bureau of Land Management, and the Department of Agriculture, Forest Service, have handbooks that prescribe guidelines for aerial and ground application operations. Regional publications, such as BLM's *Western Oregon Program—Management of Competing Vegetation Environmental Impact Statement* and the Forest Service's *Region 6 Vegetation Management Program Environmental Impact Statement*, serve to further refine herbicide application guidelines. The *Siskiyou National Forest Aerial Applicator's Handbook* (USDA 1982) is an example of a forest-level operational guideline that specifies detailed herbicide application procedures.

Aerial and ground application procedures undergo detailed planning weeks or even months in advance. Mitigation measures, such as not spraying in sensitive areas, notifying the public, posting warning signs, and conducting water monitoring, are specified in site-specific annual vegetation management plans. Many mitigation measures developed for herbicide operations in the 13 Western States are described in previous environmental impact statements, which this document supplements. Some specific examples of project mitigation measures include the following:

(1) Application operations will typically be suspended when any of the following conditions exist on the treatment area:

(a) Wind velocity exceeds 6 miles per hour (liquids), 15 miles per hour (granular), unless a lower maximum wind speed is specified on the label.

(b) Snow or ice covers the target foliage.

(c) Precipitation is occurring or is imminent.

(d) Fog significantly reduces visibility.

(e) Air turbulence (for example, thermal updrafts) is sufficient to affect the normal chemical distribution pattern.

(2) During air operations, a radio network will be maintained to link all parts of the project.

(3) Equipment will be designed to deliver a median droplet diameter of 200 to 800 microns. This droplet size is large enough to avoid excessive drift while providing adequate coverage of target vegetation.

(4) Individuals involved in the herbicide handling or application will be instructed on the safety plan and spill procedures.

References

BLM. U.S. Department of the Interior, Bureau of Land Management.

U.S. Department of Agriculture, Forest Service, Pacific Northwest Region. 1982. *Siskiyou national forest aerial applicators handbook.*

U.S. Department of the Interior, Bureau of Land Management. 1985. *Northwest area noxious weed control program, final environmental impact statement.*

Section E3
Human Health Hazard
Analysis

Introduction

This section presents the results of the hazard analysis: a review of available information on the toxicity of the 19 herbicides—amitrole, atrazine, bromacil, chlorsulfuron, clopyralid, 2,4-D, dalapon, dicamba, diuron, glyphosate, hexazinone, imazapyr, mefluidide, metsulfuron methyl, picloram, simazine, sulfometuron methyl, tebuthiuron, and triclopyr—proposed for BLM vegetation treatments.

The first subsection describes the sources of toxicity information used in the hazard analysis. The second subsection explains the terminology of laboratory toxicity testing used later in describing the herbicides' toxic properties. The third subsection presents toxicity summaries of each herbicide drawn from the available information, and it describes the potential for each to cause systemic effects, reproductive or developmental effects, cancer, and heritable genetic mutations. The details of the derivation of cancer potency are presented for those herbicides suspected of being carcinogenic. The final subsection reviews the toxicity information for the herbicide carriers diesel oil and kerosene.

Sources of Toxicity Information

The toxicity of 10 of the herbicides (amitrole, atrazine, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, picloram, simazine, and triclopyr) to laboratory animals and humans is described in detail in the background statements of the Forest Service Agricultural Handbook No. 633 (USDA 1984). Tebuthiuron toxicity is described in a background statement prepared for the Forest Service as a supplement to Handbook No. 633. The toxicity of the herbicides diuron and bromacil is described in background statements written in conjunction with the BLM western Oregon risk assessment. The toxicity of clopyralid, chlorsulfuron, mefluidide, metsulfuron methyl, and prometryn are described in detail in background statements prepared for this EIS. These documents are incorporated by reference into this risk assessment and BLM's EIS in accordance with 40 CFR 1502.16 and are available for review at all BLM District Offices

in the EIS States as well as at the address shown on the cover page.

Much of the data on pesticide toxicity have been generated to comply with the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended (7 U.S.C. 136 et seq.), which establishes procedures for the registration, classification, and regulation of all pesticides, including herbicides. The Environmental Protection Agency (EPA) is responsible for implementing FIFRA. EPA registration standards are thorough reviews of all data submitted for registration or re-registration of a chemical and are available through EPA's Freedom of Information Office. EPA has compiled "science chapters" that include discussions of toxicity on many of the herbicides (amitrole, bromacil, dicamba, diuron, hexazinone, picloram, and simazine), and these are also available from EPA. EPA compiles toxicity levels and related information from the series of studies submitted for registration in summary tables called "tox one-liners" that are available on request from its Freedom of Information Office. A large body of additional toxicity information exists in the open literature, particularly for chemicals such as 2,4-D that have been used for many years.

A literature search was funded by the Bureau of Land Management to ensure that all of the relevant available information was used in this risk analysis. The National Library of Medicine's Registry of Toxic Effects of Chemical Substances (RTECS) and Hazardous Substances Data Bank (HSDB) data bases, as well as Medline, Chem Abstracts Embase (Excerpta Medica), and International Pharmaceutical Abstract data bases were searched in 1986 to locate current literature pertaining to the carcinogenicity and mutagenicity of the herbicides. That search was updated to make the document current for information available as of January 1989.

Data from the U.S. Department of Agriculture, Forest Service, Pesticide Background Statements (USDA 1984), and the California Department of Food and Agriculture Summaries of Toxicological Data were reviewed and compared to summaries of studies submitted to EPA for registration of the 20 herbicides. Whenever possible, studies

that EPA reviewed and validated were used to set toxicity reference levels. No EPA-invalidated studies were used.

Hazard Analysis Terminology

Because of obvious limitations on the testing of chemicals on humans, judgments about the potential hazards of pesticides to humans are necessarily based on the results of toxicity tests on laboratory animals. These toxicity test results are supplemented by information on actual human poisoning incidents and effects on human populations when they are available. The discussion of laboratory toxicity testing that follows is drawn from Hayes (1982), Doull et al. (1980), and Loomis (1978).

Laboratory Toxicity Testing

Test Animal Species

Laboratory test animals function as models of the likely effects of a pesticide in humans. Ideally, the test animal should metabolize the compound the same as a human would and should have the same susceptible organ systems. Results of such tests could then be directly extrapolated to humans with some adjustment made for differences in body weight and body surface area. Although no test animal has proven ideal, a number of species have proven to be consistent indicators for certain types of toxicity tests, routes of administration, and types of chemicals; in particular, rats, mice, rabbits, hamsters, guinea pigs, dogs, and monkeys.

Toxicity Endpoints and Toxicity Reference Levels

Toxicity is the ability of a substance to produce an adverse effect on an organism. In general, adverse effects progress relative to duration of exposure. Toxicity tests are designed to identify specific toxicity endpoints, such as death or cancer, and toxicity reference levels, such as an LD_{50} or no-observed-effect level (NOEL). In addition to the test animal used (previously discussed), toxicity tests vary according to test duration, route of administration, dose levels, dosing schedule, number of test groups, and number of animals per group. Toxicity tests also vary on the basis of whether it is assumed that the effect in question is a threshold effect or a nonthreshold effect.

Threshold and Nonthreshold Effects

Most chemicals are assumed to have a threshold level of toxic effects on a local basis (at the site of administration) or systemic basis (acting throughout the body), below which no adverse effects occur to the test organism. Chemicals are generally thought to possess no such threshold level for cancer and mutations, thus these toxic endpoints may occur (with a certain level of probability) even in the presence of extremely small quantities of the substance. In the discussion of each herbicide in this hazard analysis, threshold effects are discussed first; nonthreshold effects (cancer and mutagenicity) are discussed second. The term "greater than," which is used frequently to describe threshold effect, indicates that no adverse effects have been observed at the highest dosage level.

Duration of Toxicity Tests

The duration of toxicity tests ranges from very short-term acute tests to longer subchronic studies to chronic studies that may last the lifetime of an animal. Acute toxicity studies involve administration of a single dose to each member of a test group (either at one time or in a cumulative series over a short period of less than 24 hours) or several daily doses over a short duration (with a maximum duration of 2 weeks). Subchronic toxicity studies, used to analyze the effects of multiple doses, usually last from 3 weeks to 3 months but generally last less than one-half the lifetime of the test animal. Chronic studies, also used to analyze the effects of multiple or continuous doses, normally last 2 years or more but generally more than one-half of the test species' lifetime.

Routes of Administration

Routes of administration include oral by gavage (forced into the stomach with a syringe through plastic tubing) or fed in the diet, dermal (applied to the skin), inhalation (through exposure to vapors or aerosol particles), and parenteral (injection other than into the intestine). Parenteral routes include subcutaneous (injected under the skin), intraperitoneal (injected into the abdominal cavity), and intravenous (injected into a vein). Oral, dermal, and inhalation doses most nearly duplicate the likely routes of exposure to humans; therefore, these administration routes are used most frequently in toxicity testing. In addition, ingestion and inhalation are considered the most important routes of exposure for pesticides in humans.

Dosing Levels

Doses are expressed in several ways. They can be expressed as milligrams (mg, which is 1/1,000 of a gram) of the chemical per kilogram (kg, which is 1,000 grams) of body weight of the test animal, or in parts per million (ppm) in the animal's diet, or in milligrams per liter (mg/L) in the air the animal breathes or in the water it drinks. In long-term studies, the test substance is generally administered in the diet with specified amounts in parts per million. The body weight and food consumption of the test animal over the test period is used to convert parts per million in the diet to milligrams of chemical per kilogram of body weight per day (mg/kg/day) for extrapolation to humans. In most chronic toxicity studies, at least three dosing levels are used in addition to a zero-dose, or control group. In general, the control group animals are administered the vehicle (for example, water or saline) used in administering the test material. In a dietary study, the basal feed serves as the vehicle.

Types of Laboratory Toxicity Studies Used in the Risk Assessment

Acute Toxicity Studies

Acute toxicity studies are used to determine a number of toxicity endpoints based on a single dose or several large doses of a substance. An important endpoint in acute testing is the toxicity reference level known as the median lethal dose (LD_{50}), which is the dose, usually administered orally, that kills 50 percent of the test animals. The lower the LD_{50}, the greater the toxicity of the chemical. The LD_{50} ranges for the acute oral toxicity categories used in this risk assessment are those of the EPA classification system using rat oral LD_{50}s, as shown in Table E3-1 (adapted from Maxwell 1982). Acute toxicity studies are also used to estimate dose levels to be used in longer term studies. In addition to the acute oral LD_{50} test in rats, in its battery of laboratory toxicity studies considered as acute tests, EPA (40 CFR Part 158) includes acute dermal, acute inhalation (rat), eye irritation (rabbit), dermal irritation (rabbit), dermal sensitization (guinea pig), and acute delayed neurotoxicity (hen). The last test is required for chemicals, such as organophosphate, that are known to cause cholinesterase depression or other nervous system effects. Because lethality is the intended toxic endpoint in the acute oral, dermal, and inhalation studies, dose levels usually are set relatively high. Toxic symptoms displayed by the animals may be recorded throughout the study, and tissues and

organs are examined for abnormalities at the end of the test. The animal most commonly used for oral LD_{50}s is the rat. Rabbits are used most often to determine dermal LD_{50}s.

Figure E3-1 illustrates the relationship between the LD_{50} and the dose level at which no adverse effects were observed (NOEL). For longer term tests, the adverse effects may occur on a continuum and progress in intensity. Table E3-2 summarizes the dermal toxicity of the herbicides.

Subchronic Toxicity Studies

Subchronic studies are designed to determine the effects of repeated exposure and, in particular, the toxicity reference level called the no-observed-effect level (NOEL), which is the highest dose level at which no toxic effects are observed. If a chemical produces effects at the lowest dose tested (LDT) in a study, the NOEL must be at some lower dose. If the chemical produces no effects, even at the highest dose tested (HDT), the NOEL is equal to or greater than the HDT. Another toxic endpoint of interest is the lowest dose showing toxic effects, the lowest effect level (LEL). For local and systemic effects, the chemical's effect threshold lies between the NOEL and LEL for the tested species (Figure E3-1). EPA (40 CFR Part 158) includes 90-day feeding tests (rodent and nonrodent), 21-day dermal, 90-day dermal, 90-day inhalation, and 90-day neurotoxicity studies in its battery of subchronic testing requirements under FIFRA.

Subchronic studies, normally employing lower dose levels than acute tests, provide information on systemic effects, cumulative toxicity, the latency period (the time between exposure and the manifestation of a toxic effect), the reversibility of toxic effects, and appropriate dose ranges to be used in chronic tests. Adverse effects may range from death in the extreme case to minor debilitating, often reversible, effects such as decreased rate of food consumption; changes in body weight; decreased enzyme levels; changes in blood constituents, such as red blood cells (RBCs) or white blood cells (WBCs); undesirable constituents in the urine; or microscopic changes in tissues.

Chronic Toxicity Studies

Chronic studies, like subchronic studies, are used to determine systemic NOELs. All other things being equal, the longer the study from which a NOEL is derived, the more reliable the resulting value. Chronic studies, however, are even more important in determining doses that

Table E3-1. Acute Toxicity Classification and Acute Toxicities of the 19 Herbicides and Additives Being Evaluated for Use in Vegetation Management in Relation to Other Chemicals

Toxicity Category[a] (label signal words)		Herbicide or Other Chemical Substance	Oral LD$_{50}$ for Rats (mg/kg)		Equivalent Human Dose
IV	Very slight		5,000 - 50,000	(range)	More than 1 pint
		Sugar	30,000		
		Kerosene	28,000		
		Ethyl alcohol	13,700		
		Dalapon	7,577		
		Diesel Oil	7,380		
		Chlorsulfuron	5,545		
		Imazapyr	>5,000		
		Sulfometuron Methyl	>5,000		
		Simazine	>5,000		
		Metsulfuron Methyl	>5,000		
III	Slight (caution)		500 - 5,000	(range)	1 ounce to 1 pint
		Mefluidide	>4,000		
		Glyphosate	4,320		
		Clopyralid	4,300		
		Picloram	4,012		
		Bromacil	3,998		
		Diuron	3,750		
		Table salt	3,750		
		Bleach	2,000		
		Aspirin, Vitamin E$_3$	1,700		
		Hexazinone	1,690		
		Amitrole	1,100		
		Formaldehyde	800		
		Dicamba	757		
		Atrazine	672		
		Tebuthiuron	644		
		Triclopyr	630		

[a]Categories, signal words, and LD$_{50}$ ranges are based on a classification system used by EPA for labeling pesticides. Adapted from Maxwell (1982).

Table E3-1. Acute Toxicity Classification and Acute Toxicities of the 19 Herbicides and Additives Being Evaluated for Use in Vegetation Management in Relation to Other Chemicals (continued)

Toxicity Category[a] (label signal words)	Herbicide or Other Chemical Substance	Oral LD_{50} for Rats (mg/kg)		Equivalent Human Dose
II Moderate (warning)		50 - 500	(range)	1 teaspoon to 1 ounce
	2,4-D	375		
	Caffeine	200		
I Severe (danger - poison)		0 - 50	(range)	1 teaspoon or less
	Nicotine	50		
	Strychnine (rodenticide)	30		
	Parathion (insecticide)	13		
	TCDD (a dioxin)	0.1		
	Botulinus Toxin	0.00001		

LD$_{50}$ - Acute lethal dose. One-time or short-term dose that is lethal to 50 percent of treated animals.

Threshold - Dose level at which toxic effects are first observed in test animals.

NOEL - No-observed-effect level. Long-term dose that does not result in apparent adverse effects in test animals.

LD$_{50}$

Threshold
NOEL

(Not to Scale)

Toxic effects observed -- many deaths

Observed response

Moderate toxic effects observed -- few deaths

Some toxic effects observed -- no deaths

No effects observed --

NOEL Threshold

LD$_{50}$

Low
High

Dose
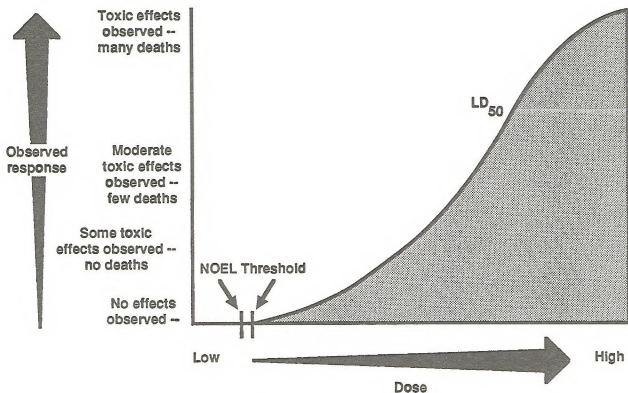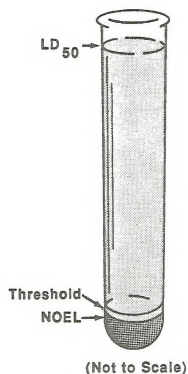
Figure E3-1. Relationships among toxicity reference levels.

Table E3-2. Dermal Toxicology Studies of the 19 Herbicides and Additives

Chemical	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
Amitrole	III,[a] LD$_{50}$ for Amizol®= 10,000 mg/kg, rabbits tested (USDA 1984).	NA[b]	III, rabbits tested (EPA 1985a).	NA
Atrazine	III, LD$_{50}$ >2,000 mg/kg, rats tested (EPA 1983).	IV, rabbits tested (EPA 1983).	II, rabbits tested (EPA 1983).	NA
Bromacil	III, LD$_{50}$ >2,000 mg/kg, rabbits tested (EPA 1986).	IV, rabbits tested (EPA 1986).	III, rabbits tested (EPA 1986).	NA
Chlorsulfuron	III, LD$_{50}$ >3,400 mg/kg, rabbits tested (Dupont 1982).	IV, 80% a.i., rabbits tested (EPA 1988b).	III, rabbits tested (Dupont 1982).	NA
Clopyralid	III, LD$_{50}$ >2,000 mg/kg, rabbits tested (WSSA 1983).	NA	NA	Slight or questionable irritation at unspecified dose levels, 14-day dermal test on rabbits (EPA 1988).
2,4-D	III,[a] LD$_{50}$ >3,980 mg/kg, 21.1% 2,4-D, rabbits tested (EPA 1986a).	III, 21.1% 2,4-D, rabbits tested (EPA 1986a).	I, rabbits tested (EPA 1986a).	No toxic symptoms at doses up to 3,980 mg/kg in rabbits (USDA 1984).
Dalapon	III, 26.8% formulation of dalapon, LD$_{50}$ >4,000 mg/kg, rabbits tested (EPA 1984).	NA	III, 26.8% formulation of dalapon, rabbits tested (EPA 1984).	NA

[a]Environmental Protection Agency labeling guidelines for pesticides applied to skin or eyes.
    I.   Irreversible corneal opacity at 7 days; corrosive to skin.
    II. Corneal opacity reversible within 7 days; severe skin irritation at 72 hours.
    III. No corneal opacity; moderate skin irritation at 72 hours.
    IV. No irritation to the eyes; mild or slight skin irritation at 72 hours.
[b]NA = Not available.

Chemical	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
Dicamba	III, LD_{50} >2,000 mg/kg (ODT), DMA salt, rabbits tested (EPA 1984).	IV, rats tested; rabbits tested (EPA 1984).	I, rabbits tested (EPA 1983).	Slight dermal irritation and edema at a 100 mg/kg/day dosage level, moderate dermal irritation and edema at a 2,500 mg/kg/day dosage level, rabbit 21-day dermal test (EPA 1984).
Diuron	III, LD_{50} >2,000 mg/kg, rabbits tested (EPA 1986).	III, rabbits tested (EPA 1983).	IV, rabbits tested (EPA 1983).	NA
Glyphosate	III, LD_{50} (F) ≥7,940 mg/kg, LD_{50} (M) ≥5,010 mg/kg, rabbits tested (EPA 1986a).	IV, rabbits tested (EPA 1986a).	III, rabbits tested (EPA 1986b).	NOEL = 1,000 mg/kg/day LEL = 5,000 mg/kg/day Slight erythema, and edema; 21-day dermal test of rabbits (EPA 1986b).
Hexazinone	III, LD_{50} >5,278 mg/kg, rabbits tested (EPA 1986).	IV, rabbits tested (EPA 1988).	I, rabbits tested (EPA 1988).	NA
Imazapyr	III, LD_{50} >2,000 mg/kg, rabbits tested; LD_{50} >2,000 mg/kg, rats tested (EPA 1985, American Cyanamid Co. 1985).	IV, rabbits tested (EPA 1985).	III, rabbits tested (EPA 1985).	NOEL = 400 mg/kg/day (HDT), 21-day dermal test on rabbits (EPA 1985).

Table E3-2. Dermal Toxicology Studies of the 19 Herbicides and Additives (continued)

Chemical	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
Light Fuel Oil	Diesel oil--III (tentative), 6 ml/kg (ODT), rabbits tested (Beck et al. 1982).	Diesel oil--II (Beck et al. 1982).	Diesel oil--IV (Beck et al. 1982).	Diesel oil--avg. weight loss of 0.38 kg/animal, no mortality; 3,280 mg/kg dosage level; 21-day dermal test of rabbits (Beck et al. 1982).
	Kerosene (Jet Fuel A)--III (tentative) LD_{50} >5,000 mg/kg, rats tested (Beck et al. 1982).	Kerosene (Jet Fuel A)--IV, rabbits tested; IV, guinea pigs tested (Beck et al. 1982).	Kerosene (Jet Fuel A)--IV, rabbits tested; IV, guinea pigs tested (Beck et al. 1982).	Kerosene (Jet Fuel A)--75% mortality (severe dermal irritation, anorexia, weight loss, depression, and pale liver and kidneys) at 6400 mg/kg/day dosage level; 21-day test of rabbits (Beck et al. 1982).
Mefluidide	III, LD_{50} >4,000 mg/kg rabbits tested (WSSA 1983).	IV, rabbits tested (WSSA 1983).	III, rabbits tested (3M 1987).	NA
Metsulfuron Methyl	III, LD_{50} >2,000 mg/kg rabbits tested (EPA 1988b).	III, 70% a.i., rabbits tested (EPA 1988b).	II, rabbits tested (Du Pont 1984).	NA
Picloram	III, LD_{50} >2,000 mg/kg (HDT), rabbits tested (EPA 1988a).	IV (tech. washed) rabbits tested (EPA 1988a).	III, rabbits tested (EPA 1988a).	NA

Chemical	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
Simazine	III, LD$_{50}$ >10,000 mg/kg, rabbits tested (EPA 1983).	NA	III, rabbits tested (USDA 1984).	NOEL = greater than 1,000 mg/kg/day, 3-week dermal test on rabbits (EPA 1983b).
Sulfometuron Methyl	III, LD$_{50}$ >2,000 mg/kg, rabbits tested (EPA 1984).	III, rabbits tested; IV, guinea pigs tested 75% a.i. (EPA 1984).	III, 75% a.i. (EPA 1984).	NOEL >2,000 mg/kg/day (HDT) OUST formulation, 21-day dermal rabbit test (EPA 1984).
Tebuthiuron	II, LD$_{50}$ >200 mg/kg, rabbits tested (EPA 1986).	IV, rabbits tested (EPA 1986).	IV, rabbits tested (EPA 1986).	NA
Triclopyr	III, LD$_{50}$ ≥2,000 mg/kg (ODT), no mortalities, rabbits tested (EPA 1986).	IV, rabbits tested (EPA 1986).	II, rabbits tested (EPA 1986).	NA

are hazardous to reproductive success or in determining whether the chemical causes cancer. EPA (40 CFR 158) includes chronic toxicity (feeding) studies (rodent and nonrodent), oncogenicity (cancer) studies (rat and mouse), teratogenicity studies (rat and rabbit), and reproduction studies in its battery of chronic testing requirements under FIFRA.

Teratogenicity Tests. Teratogenicity tests (teratology studies) are conducted to determine the potential of a chemical to cause malformations in an embryo or a developing fetus between the time of conception and birth. These studies generally use pregnant rats or rabbits dosed during the middle period of gestation while the organs of the fetus are developing. The animals are monitored for functional as well as structural deformities.

Reproduction Studies. Reproduction studies are conducted to determine the effect of the chemical on reproductive success as indicated by fertility, direct toxicity to the developing fetus, and survival and weight of offspring for low-level, long-term exposure. These tests are usually performed at lower doses than those used in teratogenicity studies and they normally use rats. Both male and female rats are exposed to the chemical for a number of weeks before mating. The number of resulting pregnancies, stillbirths, and live births are recorded. Tests may be conducted over two or three generations.

Carcinogenicity Tests. Oncogenicity is defined as the ability to induce tumors. Benign, as well as malignant tumors, are considered as evidence of potential carcinogenicity. Oncogenicity tests are conducted to determine the potential for a chemical to cause tumors when fed in the diet over the test animal's lifetime. Testing is normally conducted with rats or mice for a 2-year period.

The cancer potency of a chemical is defined as the increase in likelihood of getting cancer from a unit increase in the dose of the chemical. It should be noted that the potency is derived from data at high-dose levels; therefore, to apply the formula to low doses, one must assume the applicability of the formula. An example of this relationship is illustrated by the graph in Figure E3-2. The slope of the line specifies what the increase in cancer probability is for each unit increase in dose in milligrams per kilogram per day (mg/kg/day). The cancer potency value reflects the probability of getting cancer sometime in a person's lifetime for each mg/kg/day.

The cancer potency is derived from tumor data generated in laboratory animal studies. Note in Figure E3-2 that the dose levels used in the laboratory cancer studies are high, but those that humans are likely to experience from exposure to the environment are low. The figure also shows that the potency, in general, is a function of the applied dose. Note also that the line relating dose to cancer probability approximates a straight line in the low-dose region.

Several assumptions have been made in estimating cancer potencies. First, it is assumed that any dose, no matter how small, has some probability of causing cancer. This is an assumption based on the nonthreshold hypothesis, discussed previously, which postulates that even a single, extremely small dose may be enough to trigger cancer. Second, one of the principal areas of scientific controversy in cancer risk assessment is extrapolating the cancer potency line from the high doses used in animal studies to the far lower doses to which humans may be exposed. Models other than the linearized multistage model, which assumes a straight line at low doses, as illustrated in Figure E3-2, have been used for the extrapolation of cancer data to assess human risk. However, this model is believed to be reasonably conservative (not underestimating risk), and it is the model EPA uses. Third, the cancer potency used in calculating human risk in this analysis is not the maximum likelihood potency value, but the upper limit value of the 95-percent statistical confidence interval.

Mutagenicity Assays

This section describes how the results of mutagenicity assays were used to draw conclusions about the risk of a chemical causing genetic effects. Mutagenicity assays are used to determine the ability of a chemical to cause structural changes (mutations) in the basic genetic material (DNA) of germ cells or somatic cells. Germ cell genetic defects could possibly lead to the passing of defective genetic instructions to offspring. The offspring may develop diseases or malformations or be predisposed to diseases because of those inherited defects. Somatic cell genetic defects are believed to play a role in the development of certain diseases, in particular, cancer.

Heritable Genetic Disease. Genetic diseases and abnormal phenotypes (for example, congenital anomalies) are produced in humans as a consequence of genetic errors occurring at the gene or chromosome levels (McKusick 1983, Denniston 1983). Most humans affected
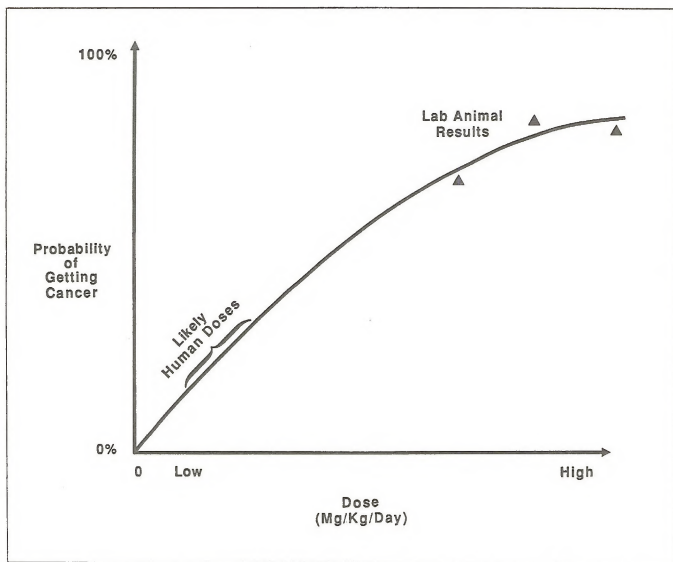
Figure E3-2. Cancer potency curve.

by genetic disease inherited their disease or predisposition for the disease as a pre-existing genetic error (Matsunaga 1982, Carter 1977). The same is true for congenital anomalies. A small percentage of affected individuals represent "new" mutations that were not pre-existing in the germ lines of their parents. The specific causes of these "new" mutations are unknown but could arise spontaneously, or could be induced by natural mutagens (for example, aflatoxins or background radiation), therapeutic regimens (cancer treatment with agents such as cytoxan or Adriomycin), or environmental or occupational exposures to mutagenic chemicals (Brusick 1987).

To date, epidemiological studies of human populations have revealed the existence of more than two dozen human carcinogens but have failed to confirm epidemiologically an agent that could be legitimately classified as a human germ cell mutagen. Consequently, assessments of human genetic risk must be built upon evidence from nonhuman sources and extrapolated to human populations.

According to EPA's guidelines for germ cell mutagenicity risk assessment (Fed. Reg. 51(185):34006-34012, Sept. 24, 1986), mutagenic endpoints of concern include point mutations (submicroscopic changes in the base sequence of DNA) and structural or numerical chromosome aberrations. Structural aberrations include deficiencies, duplications, insertions, inversions, and translocations. Numerical aberrations are gains or losses of whole chromosomes. Other relevant test endpoints include DNA damage, unscheduled DNA synthesis (UDS), recombination and gene conversion, and sister chromatid exchange (SCE).

The species used in mutagenicity assays range from primitive organisms, such as the bacteria *Salmonella*, *Escherichia*, and *Streptomyces*; the mold *Aspergillus*; the yeast *Saccharomyces*; and the fruit fly *Drosophila*, to more advanced organisms including mammalian species. Tests may be conducted in vivo (within the body of the living organism) or in vitro (on cells cultured outside the body in a petri dish or test tube).

According to Dr. David Brusick (1987), data that might be used in germ cell mutagenicity risk assessments come from mutagenicity studies that can be categorized as follows:

Mammalian germ cell tests:

   Mammalian model studies for germ cell alterations consist predominantly of tests on

rodent models (typically the mouse) for transmissible effects (specific locus, heritable translocation, and selected dominant genes) and nontransmissible effects (dominant lethal, chromosomal aberrations, gonadal DNA damage and repair).

Short-term tests:

   (1) Mammalian model studies for somatic cell alterations include many of the tests commonly used in genetic toxicology such as chromosome analysis, micronucleus tests, tests for unscheduled DNA synthesis (UDS), and measurements of DNA adducts.

   (2) Submammalian model studies for germ cell or somatic cell alterations—typical tests in this group are the *Drosophila* sex-linked recessive lethal assay and the *Salmonella* reverse mutation assay (Ames test).

   (3) Mammalian cell in vitro tests—cultured mammalian cells can be screened for all classes of genetic alterations (that is, chromosome damage, gene mutation, UDS).

Genotoxic Carcinogens. NRC (1987) states that there are two broad mechanisms by which chemicals cause cancer: by some direct chemical interaction with the DNA structures of the cell or by indirect effects on the cellular environment that increase the tumor yield without direct chemical alteration of DNA. The former are termed genotoxic carcinogens and the latter, epigenetic carcinogens.

EPA describes the use of mutagenicity tests as evidence in judging the likelihood that a chemical is a genotoxic carcinogen. According to EPA's guidelines for carcinogen risk assessment (Fed. Reg. 51(185):33992-34003, Sept. 24, 1986):

   Tests for point mutations, numerical and structural chromosome aberrations, DNA damage/repair, and in vitro transformation provide supportive evidence of carcinogenicity and may give information on potential carcinogenic mechanisms. A range of tests from each of the above end points helps to characterize an agent's response spectrum.

   Short-term in vivo and in vitro tests that can give indication of initiation and promotion activity may also provide supportive evidence for carcinogenicity. Lack of

positive results in short-term tests for genetic toxicity does not provide a basis for discounting positive results in long-term animal studies.

The methods for cancer risk analysis using animal data have been reasonably well formulated. However, in the absence of rodent cancer data or negative rodent cancer data, positive results from short-term tests for genotoxicity have been used as justification for questioning the adequacy of the rodent cancer studies. The rationale for such a use of short-term assays rests with the close mechanistic and correlative association between carcinogens and mutagens (Brusick 1987, Shelby 1988).

Estimates of cancer potency that are used to assess cancer risk are based on the results of long-term feeding studies indicating tumor induction rather than on the results of short-term mutagenicity assays. An approach that has been suggested by some experts is to develop worst-case estimates of cancer risk from cancer studies regardless of whether the studies show significant evidence of increasing tumor incidence with increasing dose. This risk assessment does not adopt this approach because the accepted practice in EPA and the scientific community is to consider only those chemicals with positive tumor evidence as potential human carcinogens.

It is assumed in regard to heritable mutagenicity risk that the cancer tests are the more sensitive toxic endpoint (that is, that no chemical that has been shown to be a germ cell mutagen has not been shown to be carcinogenic at lower doses) and this would constitute the worst-case estimator of risk.

Use of Short-Term Tests to Evaluate Germ Cell Risk

Background. The published EPA guidelines cited above for using short-term test data in assessing mutagenic risk fail to provide recommendations for establishing quantitative risk estimates. Although the EPA guidelines provide broad qualitatively descriptive risk classifications, they are insufficient for formulating a quantitative comparison of two different chemicals that may fall into the same general class. Therefore, Government agencies, such as BLM and the Forest Service, have no guidelines for conducting quantitative risk assessments to reach worst-case risk estimates, which should be at least semiquantitative.

Each type of test described above has its particular advantages and limitations. Knowledge of their advantages and disadvantages is important in extrapolating test responses to humans. There may be a tendency to use a positive response from an in vitro assay, for example, to operationally define a tested chemical as a mutagen even when the chemical is not shown to be mutagenic in any other test. This approach to hazard identification is an inappropriate use of such in vitro tests. Further extension of these limited positive findings into a presumption of genetic risk is not supported by the available scientific evidence.

Correlation of Rodent Germ Cell Tests With Short-Term Test Results. Although no chemical has been conclusively established as a human germ cell mutagen, evidence from studies showing chemical-induced mutations in human somatic cells as well as the identification of rodent germ cell mutagens argue that at least some "new" human mutations and their resultant pathologies are the consequence of environmental exposures to mutagenic chemicals. However, without human data, mammalian germ cell models (that is, mouse assays) will have to serve as the experimental standard upon which human risk estimates are based (Ehling 1988). If the logic of inferring human germ cell risk from the results of rodent germ cell tests is accepted, then one can determine the relative predictive accuracy of any of the nongerm cell tests identified in the previous section for identification of germ cell mutagens.

Three review articles have summarized the results of such an exercise (ICPEMC Committee 1, Russell et al. 1984, Bridges and Mendelsohn 1986). The scientific evidence indicates, however, that no nongerm cell test is sufficiently accurate to predict the effects that would be obtained from animal germ cell tests. Therefore, positive responses from such tests cannot be considered evidence supporting a presumption of mutagenic risk.

A Weight-of-Evidence Approach to Germ Cell Mutagenicity Risk. The next approach to the use of the abundance of nongerm cell test (that is, short-term test) results is to establish a weight-of-evidence approach for collectively evaluating the composite response from all tests conducted on a given agent.

Several qualitative (EPA 1986) and quantitative (Pet-Edwards et al. 1985, Brusick et al. 1986) weight-of-evidence schemes for mutagenicity data have been proposed. None of these weight-of-evidence schemes have been

examined in detail for concordance with the rodent germ cell data base. However, it is probably wise to use some type of weight-of-evidence scheme to evaluate short-term studies.

The only scientifically sound method for establishing human germ cell mutagenic risk is to use validated rodent models for assessing heritable gene or chromosomal mutations. The use of isolated positive responses from short-term tests (nongerm cell tests in mammals, submammalian assays, or mammalian cell in vitro tests) to establish genetic risks is not supported by available data and would be an inappropriate use of such data. In the absence of rodent germ cell data, a weight-of-evidence approach should be applied when using short-term test results to identify potential genetic hazard.

The weight-of-evidence discussion of the results of mutagenicity assays for the 20 herbicides in this risk assessment deals with those assays on the basis of three broad groups of mutagenicity endpoints: (1) tests for detecting gene mutations, (2) tests for detecting chromosomal aberrations, and (3) tests for detecting primary DNA damage.

Group 1 tests include microbial assays, involving prokaryotic (bacteria) and eukaryotic microorganisms (yeasts, fungus) developed to detect reverse mutations and, to a limited extent, forward mutations. Because many mutagens are inactive before bioactivation (by metabolic activity), bacterial tests may include a bioactivation system, such as an S9-fraction, consisting of microsomal enzymes of rats' or other animals' livers to activate the mutagen. A host-mediated assay is conducted to detect mutagenic effects in a microorganism, such as bacteria, by injecting it into the peritoneal cavity of the host (usually mice) to allow for bioactivation of the mutagen in vivo. Other tests useful for predicting gene mutations are the fruit fly sex-linked recessive lethal test, which measures the frequency of lethal mutations; the mouse specific locus test, which detects mutagenicity in germ cells in vivo; and mammalian somatic cell assays in vitro using mouse lymphoma cells, human lymphoblast, and Chinese hamster ovary cells to detect forward and reverse mutation.

Group 2 tests for detecting chromosomal effects include mammalian cytogenetic assays in Chinese hamster ovary cells in vitro and mice bone marrow micronucleus in vivo. The dominant lethal test in rodents, which determines lethal mutation in germ cells, and the heritable translocation test in mice, which

detects the heritability of chromosomal damage, are important tests performed with live animals. Fruit flies and other insects also are used to detect heritable chromosomal effects in vivo.

Group 3 tests for the existence of DNA damage caused by mutagens are based on detection of the damage by biologic processes, such as DNA repair and recombination, which occur after DNA damage. Tests to determine such processes use bacteria, yeast, and mammalian cells in vitro, with or without metabolic activation. Unscheduled DNA synthesis, for example, is often used to indicate DNA repair in human cells in vitro. Mitotic recombination and gene conversion indicate DNA damage in yeast, and sister chromatid exchange indicates DNA damage in mouse lymphoma cells, Chinese hamster ovary cells, and human lymphocytes.

The weight-of-evidence approach used in this risk assessment is similar to that of EPA (1986). It places greater emphasis on assays conducted in germ cells than in somatic cells (for detecting heritable mutations), in vivo rather than in vitro, in eukaryotes rather than prokaryotes, and in mammalian species rather than submammalian species. In vivo mammalian systems are considered to be of greater value because of their similarity to human physiology and metabolism. EPA (1986) classifies the evidence for potential human germ-cell mutagenicity as sufficient, suggestive, or limited, depending on the results of various tests performed. For instance, positive results in even one in vivo mammalian germ-cell mutation test are considered sufficient evidence for potential human mutagenicity of a specific chemical.

Epidemiology Studies

The effects on humans of exposure to chemicals in the environment can be derived from in vivo or in vitro laboratory studies (as described above), reports of clinical observations of isolated exposed individuals (human poisoning incidents), experimental studies in humans, or from direct observations of exposed human populations. The data on humans generally fall into two categories: clinical data on individuals and epidemiological data revealing patterns of disease or death in groups of humans exposed to single agents or to several different substances (NRC 1986). Thus, epidemiology studies are done to investigate the causes of disease in specified human populations by examining relationships between the incidence of particular disease types and factors associated with the disease,

such as the use of particular substances in the work-place. One such association is with agricultural workers' use of various pesticides and the incidence of several types of cancer among them.

Studies the National Cancer Institute conducted have found that fewer farmers die from cancer than would be expected based on the cancer death rate among the general population in the United States. However, farmers have a higher risk of developing lymphatic and blood-related cancer, including leukemia and cancer of the prostate, skin, and stomach (Blair 1982; Blair et al. 1985; Blair and Thomas 1979; Blair and White 1981, 1985; Cantor 1982; Cantor and Blair 1984; Weininger et al. 1987).

Although no single agricultural factor has been consistently associated with increased rates of specific cancer types, correlations with insecticide and herbicide use were noted in a number of studies (Blair and White 1985, Cantor 1982, Cantor and Blair 1984, Cantor et al. 1985). In the United States, farmers have a much lower rate of lung cancer than the general population, primarily because of their lower smoking rate (Blair 1982). However, a cohort study of pesticide-exposed agricultural workers in the German Democratic Republic (Barthel 1981) found that they had a significantly higher mortality rate from lung cancer than the general population.

In a cohort study of licensed pesticide applicators in Florida, excessive deaths were observed for leukemia and cancers of the brain and lung (Blair et al. 1983). The risk of lung cancer rose with the number of years licensed (Blair et al. 1983). Other studies have found little or no correlation between cancer incidence and pesticide use (Blair and Thomas 1979, Blair and White 1981), although factors such as exposure to oncogenic animal viruses have been related to increases in certain types of cancer (Blair 1982, Blair et al. 1985).

Animal Metabolic Elimination Studies

The herbicides evaluated in this risk assessment are rapidly excreted when administered to animals. Elimination of 90 percent or more, within 2 hours to 5 days, was reported for most of the 19 herbicides. For example, 93 percent of 2,4-D was excreted in rats within 2 hours (Grissom et al. 1985), and 100 percent was excreted within 5 days (Fisher et al. 1985). Dicamba studies revealed up to 100 percent excretion within 48 hours in rats and 99 percent excretion within 4 days in

mice (EPA 1984). For glyphosate, approximately 92 percent of the dose was excreted from rabbits within 5 days (USDA 1984). Ninety-three percent of hexazinone was excreted from rats within 24 hours, and 94.2 to 100 percent was excreted within 72 hours (USDA 1984). Picloram excretion was 90 percent within 48 hours for dogs (USDA 1984) and 96 percent within 24 hours for an unspecified animal (Nolan et al. 1984, as cited in Lavy and Mattice 1986). Eighty-three to ninety-one percent of triclopyr was excreted from rats within an unspecified time (USDA 1984). In addition to the rapid elimination of the herbicides, tissue retention studies showed low residue concentrations in animal tissues (USDA 1984). The elimination rates are summarized in Table E3-3.

Based on the high elimination rates and low tissue retention, the 19 herbicides used for vegetation management present a low risk for bioaccumulation. Bioaccumulation analyses were therefore not conducted for this risk assessment.

Toxicity of the 19 Herbicides

Overview of Toxicity

The toxicity reference levels used in this risk assessment to describe acute and chronic threshold effects of the 19 herbicides are presented in Table E3-4. The LD_{50}s in this table are from rat oral studies. Two types of NOELs are given in Table E3-4. The first NOEL is for general systemic effects, such as growth retardation, decreased red blood cell counts, and increased thyroid weight. For amitrole, dicamba, imazapyr, picloram, tebuthiuron, and triclopyr, subchronic study NOELs were used because they are the lowest NOELs found in the literature. The second NOEL is for reproductive and developmental effects, including infertility, miscarriage, general fetal toxicity, and birth defects (teratogenicity). Where information is available, NOELs are given for reproductive and teratogenic effects. All the NOELs used are the lowest found in EPA-validated studies.

The following subsections summarize the most relevant acute, subchronic, and chronic toxicity tests conducted on the 19 herbicides. These studies are included under the "Threshold Effects" subsection of each herbicide. Areas where no validated studies exist or for which EPA has requested additional studies are noted.

Table E3-3. Elimination Rates of the 19 Herbicides

Chemical	Test Animal	Elimination Rate
Amitrole	Rat	70.0 to 95.5% within 24 hours (Fang et al 1964, 1966 as cited in USDA 1984).
Atrazine	Rat	65.5% within 72 hours (Bakke et al. 1972 cited in USDA 1984).
Bromacil	NA	NA
Chlorsulfuron	Rat	95% within an unspecified period (EPA 1988b).
Clopyralid	NA	NA
2,4-D	Rat	93% within 2 hours (Grissom et al. 1985).
	Rat	100% within 5 days (Fisher et al. 1985).
Dalapon	Dog	50% within an unspecified period (Hoerger 1969 cited in EPA 1987).
	Human	50% within an unspecified period (Hoerger 1969 cited in EPA 1987).
Dicamba	Rat	100% within 48 hours (EPA 1984).
	Mouse	99% within 4 days (EPA 1984).
Diuron	NA	NA
Glyphosate	Rabbit	92% within 5 days (USDA 1984).
	Rat	94% within 5 days (FAO/WHO 1986).
Hexazinone	Rat	93% within 24 hours (USDA 1984).
	Rat	94.2 to 100% within 72 hours (USDA 1984).
Imazapyr	Rat	87% within 24 hours (American Cyanamid 1985).
Mefluidide	Cow	88.6% within 5 days (Ivie 1980).
	Sheep	83.2% within 5 days (Ivie 1980).
Metsulfuron Methyl	Rat	90.2% within 3 days (EPA 1988b).
Picloram	Dog	90% within 48 hours (USDA 1984).
	Unspecified	96% within 24 hours (Nolan et al. as cited in Lavy and Mattice 1986).
Simazine	Goat	67 to 77% within an unspecified period (Bakke and Robbins 1968 cited in EPA 1987c).
	Sheep	67 to 77% within an unspecified period (Bakke and Robbins 1968 cited in EPA 1987c).
Sulfometuron Methyl	NA	NA
Tebuthiuron	Mouse	Greater than 85% within 96 hours (Elanco Products Co. 1983).
	Rat	Greater than 85% within 96 hours (Elanco Products Co. 1983).
	Rabbit	Greater than 85% within 96 hours (Elanco Products Co. 1983).
	Dog	Greater than 85% within 96 hours (Elanco Products Co. 1983).
	Duck	Greater than 85% within 96 hours (Elanco Products Co. 1983).
	Steer	Greater than 85% within 96 hours (Elanco Products Co. 1983).
Triclopyr	Rat	83 to 91% within an unspecified period (USDA 1984).

*Not available.

Table E3-4. Laboratory-Determined Toxicity Levels Used in the Risk Analysis

Herbicide	Acute Oral LD$_{50}$ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Amitrole	1,100 mg/kg (USDA 1984).	0.5 ppm (0.025 mg/kg/day) subchronic rat feeding study (EPA 1983a, 1985b).	No birth defects observed in any studies. Fetotoxic NOEL = 4 mg/kg/day; rabbit teratology study (CDFA 1984).
Atrazine	672 mg/kg (EPA 1987).	15 ppm (0.38 mg/kg/day) 2-year dog feeding study (EPA 1988).	No birth defects in two studies. Two-generation reproductive NOEL of 10 ppm (0.5 mg/kg/day), rat (EPA 1988).
Bromacil	3,998 mg/kg (EPA 1986).	250 ppm (6.25 mg/kg/day), 2-year dog feeding study (EPA 1986, CDFA 1986).	No teratogenic effects in two studies. Greater than 12.5 mg/kg/day, three-generation rat reproduction study (EPA 1986).
Chlorsulfuron	5,545 mg/kg (Du Pont 1982).	100 ppm (5 mg/kg/day) 2-year rat feeding study (EPA 1988b).	Reproductive and maternal NOEL = 500 ppm (25 mg/kg/day), three-generation rat reproduction study (EPA 1988b). No teratogenic effects in two studies.
Clopyralid	4,300 mg/kg (WSSA 1983).	15 mg/kg/day, 2-year rat feeding study (EPA 1988).	Maternal NOEL = 75 mg/kg/day (EPA 1988). No teratogenic effects in two studies.
2,4-D	375 mg/kg (EPA 1986a).	1.0 mg/kg, first year results from 2-year rat feeding study (EPA 1985a).	Maternal NOEL = 5 mg/kg/day (EPA 1986b).
Dalapon	7,577 mg/kg (EPA 1984).	8 mg/kg/day, 2-year rat feeding study (EPA 1987).	12.5 mg/kg/day, one-generation dog reproduction study (EPA 1984).
Dicamba	757 mg/kg (USDA 1984).	15.8 mg/kg/day, subchronic rat feeding study (EPA 1987).	No teratogenic effects reported in four studies. Fetotoxic and maternal NOEL = 3.0 mg/kg/day, rabbit teratology study (EPA 1984).

Herbicide	Acute Oral LD$_{50}$ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Diuron	3,750 mg/kg (EPA 1984).	25 ppm (0.625 mg/kg/day) 2-year dog feeding study (EPA 1983).	NOEL greater than 125 ppm, three-generation rat reproduction study (6.25 mg/kg/day) (EPA 1984).
Glyphosate	4,320 mg/kg (EPA 1986a).	Greater than 31 mg/kg/day, 26-month rat feeding study (EPA 1988).	10 mg/kg/day, three-generation rat reproduction study (EPA 1986a).
Hexazinone	1,690 mg/kg (EPA 1986).	200 ppm (10 mg/kg/day), 2-year rat feeding/oncogenic study (EPA 1988).	1,000 ppm (50 mg/kg/day), three-generation rat reproduction study (EPA 1988). 125 mg/kg (HDT), rabbit teratology study (EPA 1988).
Imazapyr	Greater than 5,000 mg/kg (EPA 1985).	500 mg/kg/day (HDT), 90-day rat feeding study (American Cyanamid 1985).	Maternal toxic NOEL = 300 mg/kg/day, rat teratology study (EPA 1985b).
Mefluidide	Greater than 4,000 mg/kg (WSSA 1983).	1.5 mg/kg/day, 1-year dog feeding study (EPA 1988).	Teratogenic NOEL = 60 mg/kg/day, rat and rabbit studies (EPA 1988).
Metsulfuron Methyl	Greater than 5,000 mg/kg (EPA 1988b).	500 ppm (1.25 mg/kg/day), 1-year dog feeding study (EPA 1988b).	Maternal toxic NOEL = 25 mg/kg/day; reproductive and fetotoxic NOEL = 250 mg/kg/day (HDT), rat two-generation reproduction study (EPA 1988a).
Picloram	4,012 mg/kg, rat (EPA 1988a).	7 mg/kg, 6-month dog feeding study (Mullison 1985, EPA 1988a).	1,000 ppm (50 mg/kg/day), three-generation rat reproduction study (EPA 1988a).
Simazine	Greater than 5,000 mg/kg (EPA 1987b).	5 mg/kg/day, dog feeding study (EPA 1983).	Maternal toxic NOEL = 5.0 mg/kg/day, rabbit teratology study (EPA 1987b).

Table E3-4. Laboratory-Determined Toxicity Levels Used in the Risk Analysis (continued)

Herbicide	Acute Oral LD$_{50}$ in Rats	Lowest Systemic NOEL	Lowest Reproductive and/or Teratogenic NOEL
Sulfometuron Methyl	Greater than 5,000 mg/kg (EPA 1984).	2.5 mg/kg/day, 2-year rat feeding study (Du Pont 1986).	Reproductive NOEL = 25 mg/kg/day, two-generation rat reproduction study (Du Pont 1986).
Tebuthiuron	644 mg/kg (USDA 1984).	500 ppm (12.5 mg/kg/day), 90-day dog feeding study (EPA 1986).	Maternal toxic NOEL = 5 mg/kg/day, two-generation rat reproduction study (EPA 1987).
Triclopyr	630 mg/kg (EPA 1986).	2.5 mg/kg/day (HDT), 6-month dog feeding study (40 CFR Part 180, 50 (84):184-85, May 1, 1985).	Fetotoxic NOEL = 10 mg/kg/day, rabbit teratology study (EPA 1986).

Note: Conversion Factors:
Mouse 1 ppm = 0.150 mg/kg/day
Rat (lifetime) 1 ppm = 0.05 mg/kg/day
Rabbit 1 ppm = 0.030 mg/kg/day
Dog 1 ppm = 0.025 mg/kg/day

Source: USDA 1984.

The results of cancer and mutagenicity tests are discussed for each herbicide under the "Nonthreshold Effects" subsection. Table E3-5 summarizes the EPA-reviewed mutagenicity tests on each of the 19 herbicides for each category of testing recommended by EPA in their guidance documents on mutagenicity (EPA 1978, EPA 1984). Table E3-5 also presents the relevance of the recommended tests to a determination of human mutagenic potential according to Dr. David Brusick (1987). The weight-of-evidence approach described previously is used to assess mutagenicity risk. In general, mutagenic assays most relevant for determining heritable mutations are in vivo cell studies and germ cell or gonadal studies (for example, the mouse specific locus test). A germ cell study may be considered relevant to evaluating the germ-cell mutagenicity of a chemical even if the test organism is not mammalian (*Drosophila* sex-linked recessive lethal assay). In vitro studies using mammalian cells are less reliable because of the high percentage of false positive findings caused by nonphysiologic treatment conditions and other phenomena. Tests used to detect primary DNA damage (Group 3 in Table E3-5) are not generally reliable for determining the mutagenic potential of a chemical to affect human germ cells. Most tests reviewed in the present evaluations were derived from those reviewed by EPA in tox one-liners or EPA science chapters. If tox one-liners or science chapters were unavailable, studies of mutagenicity were obtained from USDA pesticide background statements, which reported studies from the open literature. Results reported within the same study for different test species or different test types were counted as individual tests. Therefore, a single study reported in EPA tox one-liners may be represented more than once in Table E3-5. For instance, one study that reported positive results in the Ames reverse mutation test for bacteria *Salmonella* spp. and *E. coli*, activated and inactivated, would represent four positive results in Category 1A. Males and females, as well as different strains of the same species, were counted as one test only, unless different results were reported for each.

Overall results of mutagenicity testing not subclassified into nongerm cell or germ cell assays (numbers of positive and negative assays) for each herbicide are listed in Table E3-6. For some of the herbicides, no validated mutagenicity tests exist or the mutagenicity tests conducted are insufficient to conclude whether the chemical is mutagenic. For these herbicides, the worst case analysis presented in Section E5 assumed that these herbicides are mutagenic to somatic cells. In such cases, the results of carcinogenicity tests (Table E3-6) were used to estimate mutagenic risk, based on a high correlation between mutagenic and carcinogenic activity reported in several studies (Blackburn et al. 1984; Pogodina et al. 1984; Parodi et al. 1981, 1982, 1983a,b).

The results of studies examining the ability of these herbicides to cause cancer are also discussed below and are summarized in Table E3-6. Data gaps and areas of uncertainty of all chemicals are presented following the 19 herbicide discussions. In addition, data gaps are presented in Table E3-7. For those herbicides for which a cancer risk analysis is done, the value used for cancer potency and the study from which it was derived are presented.

Amitrole

Toxic Effects Seen In Humans

Amitrole is classified by EPA as a B2 (probable human) carcinogen (EPA 1985a). Therefore, the human reference dose (or acceptable daily intake (ADI)) for chronic oral intake established by EPA (1989) is 0 mg/kg/day. The human reference dose established by the World Health Organization is 0.00003 mg/kg/day (EPA 1989).

An epidemiology study (Axelson 1974, as cited in EPA 1985a) found excess tumor incidence and mortality among Swedish railway workers exposed to amitrole. However, EPA (1985a) deemed the study inconclusive because the workers also were exposed to phenoxy acids during the same time period. A follow-up study concluded that the causal relationship of increased cancer incidence to specific pesticides remains unclear (Axelson 1980, as cited in EPA 1985a).

Threshold Toxicity In Laboratory Animals

Acute and Subacute Toxicity. Amitrole is considered to be slightly to very slightly toxic for acute effects based on LD_{50} values in rats which range from 1,100 to 25,000 mg/kg (USDA 1984). The LD_{50} for Amizol® (formulated amitrole) applied to the shaved skin of rabbits was 10,000 mg/kg (USDA 1984). Amitrole is slightly irritating to rabbits' eyes (Toxicity Category III) (EPA 1984a). Symptoms of acute toxicity in several species include intestinal paralysis, pulmonary edema, and hemorrhages in various organs (Hayes 1982).

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Amitrole	Atrazine	Bromacil	Chlorsulfuron	Clopyralid
Group 1—Tests for detecting gene mutations.						
A. Bacteria with and without metabolic activation (includes Ames assay).	+	2(+) 56(-)	2(+) 8(-)	1(+) 3(-)1(-)	1(-)	—
B. Eukaryotic microorganisms with and without metabolic activation (includes yeast assay).	+	—	4(+)	2(-)	—	—
C. Insects (e.g., sex-linked recessive lethal test).	++	3(-)	1(+) 1(-)	1(+) 1(-)	—	
D. Mammalian somatic cells in culture with and without metabolic activation (includes mouse lymphoma assay).	++	—	1(+)	—	1(-)	
E. Mouse-specific locus test in vivo.	++	—	—	—	—	
F. Mammalian germ cells in culture with and without metabolic activation.[c]	—	—	—	—	—	—
G. In vivo host mediated assay.	—	—	—	—	—	1(-)
Group 2—Tests for detecting chromosomal aberrations.						
A. Cytogenetic tests in mammals in vivo (includes rat bone marrow cell assay).	++	2(-)	2(+)	1(-)	—	1(-)

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil (continued)

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Amitrole	Atrazine	Bromacil	Chlorsulfuron	Clopyralid
B. Insect tests for heritable chromosomal effects in vivo.	++	—	—	—	—	—
C. Dominant-lethal effects in rodents, heritable translocation tests in rodents, and in vitro cytogenetic assays in mammals.	++	4(+)	1(+) 3(-)	1(-) 2(-)	—	1(-)+ +
Group 3—Tests for detecting primary DNA damage.						
A. DNA repair in bacteria (including differential killing of DNA repair defective strains and recombination assay) with and without metabolic activation.	N/A	—	2(-)	1	1(-)	—
B. Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation.	N/A	—	1(+)	1(-)	—	—
C. Mitotic recombination and gene conversion in yeast, with and without metabolic activation.	N/A	—	3(+) 6(-)	1(-)	—	—
D. Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation.	N/A	2(-)	1(-)	—	—	—

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil (continued)

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	2,4-D	Dalapon	Dicamba	Diesel Oil	Diuron	Glyphosate
Group 1—Tests for detecting gene mutations.							
A. Bacteria with and without metabolic activation (includes Ames assay).	+	12(-) 1(+)	1(-)	3(-)	2(-)	6(-)	3(-)
B. Eukaryotic microorganisms with and without metabolic activation (includes yeast assay).	+	2(-)	1(-)	—	—	—	1(-)
C. Insects (e.g., sex-linked recessive lethal test).	++	1(-) 1(+)	—	—	—	—	—
D. Mammalian somatic cells in culture with and without metabolic activation (includes mouse lymphoma assay).	++	—	—	—	2(-)	—	1(-)
E. Mouse-specific locus test in vivo.	++	—	—	—	—	—	—
F. Mammalian germ cells in culture with and without metabolic activation.[c]	—	—	1(-)	—	—	1(-)	—
G. In vivo host mediated assay.	—	4(-)	—	—	—	—	—
Group 2—Tests for detecting chromosomal aberrations.							
A. Cytogenetic tests in mammals in vivo (includes rat bone marrow cell assay).	++	—	—	1(+)	1(+)	1(-)	—

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil (continued)

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	2,4-D	Dalapon	Dicamba	Diesel Oil	Diuron	Glyphosate
B. Insect tests for heritable chromosomal effects in vivo.	++	2(-)	—	—	—	—	—
C. Dominant-lethal effects in rodents, heritable translocation tests in rodents, and in vitro cytogenetic assays in mammals.	++	3(-) 2(+)	—	1(-)	—	1(-)	—
Group 3—Tests for detecting primary DNA damage.							
A. DNA repair in bacteria (including differential killing of DNA repair defective strains and recombination assay) with and without metabolic activation.	N/A	4(-) 1(+)	—	2(+)	—	—	—
B. Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation.	N/A	2(-) 1(+)	—	1(-)	—	1(-)	1(-)
C. Mitotic recombination and gene conversion in yeast, with and without metabolic activation.	N/A	—	—	1(-)	—	—	—
D. Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation.	N/A	1(-) 1(+)	—	—	—	—	—

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil (continued)

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Hexazinone	Imazapyr	Mefluidide	Metsulfuron Methyl	Picloram
Group 1—Tests for detecting gene mutations.						
A. Bacteria with and without metabolic activation (includes Ames assay).	+	1(-)	1(-)	3(-)	1(-)	1(+) 3(-)
B. Eukaryotic microorganisms with and without metabolic activation (includes yeast assay).	+	—	—	—	—	—
C. Insects (e.g., sex-linked recessive lethal test).	++	—	—	—	—	—
D. Mammalian somatic cells in culture with and without metabolic activation (includes mouse lymphoma assay).	++	—	—	1(-)	—	—
E. Mouse-specific locus test in vivo.	++	—	—	—	—	—
F. Mammalian germ cells in culture with and without metabolic activation.	—	1(-)	—1(-)	—	1(-)	—
G. In vivo host mediated assay.	—	—	—	—	—	—
Group 2—Tests for detecting chromosomal aberrations.						
A. Cytogenetic tests in mammals in vivo (includes rat bone marrow cell assay).	++	1(-)	—	—	1(-)	1(-)

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Hexazinone	Imazapyr	Mefluidide	Metsulfuron Methyl	Picloram
B. Insect tests for heritable chromosomal effects in vivo.	++	—	—	—		—
C. Dominant-lethal effects in rodents, heritable translocation tests in rodents, and in vitro cytogenetic assays in mammals.	++	1(+)	2(-)	—	1(+)	—
Group 3—Tests for detecting primary DNA damage.						
A. DNA repair in bacteria (including differential killing of DNA repair defective strains and recombination assay) with and without metabolic activation.	N/A	—	—	1(-)	1(-)	—
B. Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation.	N/A	1(-)	1(-)	—	—	—
C. Mitotic recombination and gene conversion in yeast, with and without metabolic activation.	N/A	—	—	—	—	—
D. Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation.	N/A	—	—	1(-)	—	—

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil (continued)

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Simazine	Sulfometuron Methyl	Tebuthiuron	Triclopyr
Group 1—Tests for detecting gene mutations.					
A. Bacteria with and without metabolic activation (includes Ames assay).	+	6(-)	1(-)	3(-)	3(-)
B. Eukaryotic microorganisms with and without metabolic activation (includes yeast assay).	+	—	—	—	—
C. Insects (e.g., sex-linked recessive lethal test).	++	1(+)	—	—	—
D. Mammalian somatic cells in culture with and without metabolic activation (includes mouse lymphoma assay).	++	1(-)	—	1(+) 1(-)	—
E. Mouse-specific locus test in vivo.	++	—	—	—	—
F. Mammalian germ cells in culture with and without metabolic activation.[c]	—	—	1(-)	—	—
G. In vivo host mediated assay.	—	—	—	—	1(-)
Group 2—Tests for detecting chromosomal aberrations.					
A. Cytogenetic tests in mammals in vivo (includes rat bone marrow cell assay).	++	—	—	—	1(-)

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
+ = Applicable.
++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-5. Mutagenicity Testing on the 19 Herbicides and Light Fuel Oil (continued)

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Simazine	Sulfometuron Methyl	Tebuthiuron	Triclopyr
B. Insect tests for heritable chromosomal effects in vivo.	++	1(-)	—	—	—
C. Dominant-lethal effects in rodents, heritable translocation tests in rodents, and in vitro cytogenetic assays in mammals.	++	—	1(-)	1(-)	1(+) 1(-)
Group 3—Tests for detecting primary NA damage.					
A. DNA repair in bacteria (including differential killing of DNA repair defective strains and recombination assay) with and without metabolic activation.	NA	—	—	—	—
B. Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation.	NA	2(-) 1(+)	1(-)	1(-)	—
C. Mitotic recombination and gene conversion in yeast, with and without metabolic activation.	NA	—	—	—	—
D. Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation.	NA	1(-)	—	—	—

[a]Source: FIFRA, Environmental Protection Agency: Proposed guidelines for registering pesticides in the U.S.—Hazard evaluation: humans and domestic animals. Federal Register 43:37335-37403, August 22, 1978.
[b]NA = Not applicable.
 + = Applicable.
 ++ = Greater applicability.
[c]This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

Table E3-6. Summary of Mutagenicity and Carcinogenicity of Pesticides

Herbicide	Mutagenicity	Oncogenic Results from Chronic Studies
Amitrole	Nonmutagenic 63/69 assays (USDA 1984). Does not present potential for heritable genetic effects (EPA 1985a).	A probable human carcinogen (EPA 1985a).
Atrazine	Mutagenic in 16/34 assays (USDA 1984).	Oncogenic in 1/3 studies (EPA 1988b; USDA 1984). A possible human carcinogen.
Bromacil	Nonmutagenic in 11/13 assays (EPA 1987).	Oncogenic in 1/2 studies; existing studies adequate (EPA 1986; EPA 1985). A possible human carcinogen.
Chlorsulfuron	Nonmutagenic in 5 assays (DuPont 1982).	Nononcogenic in two 2-year feeding studies accepted by EPA (1988b).
Clopyralid	Nonmutagenic in 4 assays (EPA 1988).	Nononcogenic in two 2-year feeding studies with rats and a 2-year feeding study with mice. The mouse study was rated supplementary by EPA (1988).
2,4-D	Nonmutagenic in 32/43 assays (Newton and Dost 1984; Turkula and Jalal 1985; Turkula and Jalal 1987; Basrur et al. 1976).	Oncogenic in 1/3 studies (Hansen et al. 1971; Hazelton Laboratories 1986).
Dalapon	Nonmutagenic in 3 assays (CDFA 1986).	Nononcogenic in 3 studies (USDA 1984; CDFA 1986).
Dicamba	Nonmutagenic in 6/8 assays (USDA 1984).	Nononcogenic in 1 study accepted by EPA (1988).
Diuron	Nonmutagenic in 8/9 assays (EPA 1983 and EPA 1987).	Nononcogenic in 3 studies (EPA 1983); Studies not adequate according to EPA (1983).
Glyphosate	Nonmutagenic in 8 assays (EPA 1988).	Equivocal evidence of oncogenicity in mice EPA (1986b).
Hexazinone	Nonmutagenic in 4/5 test systems (EPA 1986 and USDA 1984).	Nononcogenic in 2 studies (USDA 1984).

Table E3-6. Summary of Mutagenicity and Carcinogenicity of Pesticides (continued)

Herbicide	Mutagenicity	Oncogenic Results from Chronic Studies
Imazapyr	Nonmutagenic in 5 assays (American Cyanamid 1985, 1986).	Nononcogenic in 1 study (Biodynamics undated).
Mefluidide	Nonmutagenic in 6 assays (EPA 1988).	Nononcogenic in 2 studies (EPA 1988).
Metsulfuron Methyl	Nonmutagenic in 4/5 assays (Du Pont 1984).	Nononcogenic in 2 studies (EPA 1988b).
Picloram	Nonmutagenic in 4/5 assays (EPA 1988b; CDFA 1986).	Oncogenic in 1/2 studies (EPA 1988b).
Simazine	Mutagenic in 4/13 studies (EPA 1987a; 1989; CDFA 1986).	Oncogenic in 1 study (EPA 1989).
Sulfometuron Methyl	Nonmutagenic in 4 assays (EPA 1984 and Du Pont 1986).	Nononcogenic in 2 studies (Du Pont 1986).
Tebuthiuron	Nonmutagenic in 5/6 studies (EPA 1986).	Nononcogenic in 2 studies (EPA 1986).
Triclopyr	Nonmutagenic in 7/8 bacterial and cytogenetic assays (EPA 1986).	Nononcogenic in 2/3 studies (EPA 1986; Dow 1987; 40 CFR Part 180 50(84):184-85, May 1, 1985).

Table E3-7. Herbicide Data Gaps

Data Gaps	Amitrole	Atrazine	Bromacil	Chlorsulfuron	Clopyralid	2,4-D	Dalapon	Dicamba
Acute Testing								
Acute oral—rat	C	C	C	C	C	C	X	C
Acute dermal	C	C	C	C	C	C	X	C
Acute inhalation—rat	C	C	C	C	C	C	X	C
Eye irritation—rabbit	W	C	C	C	C	X	X	C
Dermal irritation—rabbit	W	C	C	C	C	X	X	C
Dermal sensitization—guinea pig	W	C	C	C	C	C	X	C
Subchronic testing								
90-day feeding—rodent	C	C	C	C	C	C	X	C
90-day feeding—nonrodent	C	C	C	C	C	C	X	C
21-day dermal	C	C	C	C	C	X	X	X
90-day dermal	W	C	C	C	C	C	C	C
90-day inhalation	W	C	C	C	C	C	C	C
90-day neurotoxicity	C	C	C	C	C	C	C	C
Chronic testing								
Chronic—dog	C	R	C	C	C	X	R	C
Chronic—rodent	C	C	C	C	C	C	R	C
Oncogenicity—rat	C	R	C	C	C	R	X	C
Oncogenicity—mouse	C	C	C	C	C	P	R	X
Teratogenicity—rat	P	C	C	C	C	C	X	C
Teratogenicity—rabbit	P	C	C	C	C	X	R	C
Reproduction—rat	C	R	C	C	C	C	X	C
Mutagenicity	C	R	C	C	X	R	R	C

Note: According to FIFRA Guidelines, EPA has requested the following additional toxicology information on BLM/Region 6 Forest Service herbicides.
C = Data requirement complete.
X = Data gap.
R = Required for EPA registration purposes; however, data were available for risk analysis.
W = Requirement waived.
P = Partial data gap.

Data Gaps	Diuron	Glyphosate	Hexazinone	Imazapyr[1]	Mefluidide[1]	Metsulfuron Methyl	Picloram
Acute Testing							
Acute oral—rat	C	C	P	C	C	C	C
Acute dermal	C	C	P	C	C	C	C
Acute inhalation—rat	X	C	P	C	C	C	C
Eye irritation—rabbit	C	C	C	C	C	C	C
Dermal irritation— rabbit	C	C	C	C	C	C	C
Dermal sensitization— guinea pig	X	C	C	C	C	C	C
Subchronic testing							
90-day feeding—rodent	C	C	C	C	C	C	C
90-day feeding—nonrodent	C	C	C	C	C	C	C
21-day dermal	C	C	X	C	C	C	X
90-day dermal	C	C	C	C	C	C	C
90-day inhalation	C	C	C	C	C	C	C
90-day neurotoxicity	C	C	C	C	C	C	C
Chronic testing							
Chronic—dog	C	C	X	X	C	C	R
Chronic—rodent	C	C	C	X	C	C	C
Oncogenicity—rat	X	X	C	C	C	C	X
Oncogenicity—mouse	X	X	C	C	C	C	X
Teratogenicity—rat	X	C	C	C	C	C	X
Teratogenicity—rabbit	X	C	C	C	C	C	X
Reproduction—rat	X	C	C	X	C	C	R
Mutagenicity	X	C	C	C	C	C	X

[1]No evaluation of EPA data base is currently available.
X = Data gap.
C = Data requirement complete.
R = Required for EPA registration purposes; however, data were available for risk analysis.
W = Requirement waived.
P = Partial data gap.

Table E3-7. Herbicide Data Gaps (continued)

Data Gaps	Simazine	Sulfometuron Methyl[1]	Tebuthiuron	Triclopyr[1]
Acute Testing				
Acute oral—rat	C	C	X	C
Acute dermal	C	C	X	C
Acute inhalation—rat	W	X	C	C
Eye irritation—rabbit	C	C	X	C
Dermal irritation—rabbit	C	C	X	C
Dermal sensitization—guinea pig	C	X	X	C
Subchronic testing				
90-day feeding—rodent	C	C	C	C
90-day feeding—nonrodent	C	C	C	
21-day dermal	C	X	C	C
90-day dermal	C	X	C	C
90-day inhalation	C	X	C	C
90-day neurotoxicity	C	X	C	C
Chronic testing				
Chronic—dog	C	C	C	C
Chronic—rodent	C	C	X	C
Oncogenicity—rat	C	C	X	C
Oncogenicity—mouse	C	X	X	C
Teratogenicity—rat	R	C	X	C
Teratogenicity—rabbit	X	C	X	C
Reproduction—rat	X	C	C	C
Mutagenicity	X	C	X	C

[1]No evaluation of EPA data base is currently available.
X = Data gap.
C = Data requirement complete.
R = Required for EPA registration purposes; however, data were available for risk analysis.
W = Requirement waived.

E3-34

BLM Draft Vegetation Treatment EIS

Subchronic and Chronic Toxicity. Subchronic studies indicate that technical amitrole in the diet has an antithyroid effect in laboratory rats. Enlarged thyroid glands and reduced uptake of iodine were observed at the lowest effect level of 2 ppm (0.1 mg/kg/day) in a subchronic rat feeding study. The NOEL for this study was 0.5 ppm (0.025 mg/kg/day) (EPA 1985a) and is the NOEL used in this risk assessment.

In another subchronic feeding study, male rats were fed 0, 30, 100, and 300 ppm for 4 weeks, followed by 4 weeks on the control diet. The study was designed to demonstrate the reversibility of the antithyroid effects of amitrole. At 100 ppm (5 mg/kg/day), rats showed reduced levels of T_3 and T_4. However, T_3 and T_4 values returned to control levels 3 weeks after removing amitrole from the diet. The NOEL for this study was 30 ppm (1.5 mg/kg/day) (EPA 1985a). Chronic studies include a 106-day rat study with a NOEL less than 50 ppm (2.5 mg/kg/day) and a lifetime feeding study in hamsters with a systemic NOEL of 10 ppm (1.2 mg/kg/day) (EPA 1984b).

Reproductive and Developmental Toxicity. In a two-generation reproduction study, groups of male and female rats (F_0) were fed 500 ppm (25 mg/kg/day) and 1,000 ppm (50 mg/kg/day) amitrole for 107 to 110 days (EPA 1985a). Two other groups were fed 25 (1.25 mg/kg/day) and 100 ppm (5 mg/kg/day) for 240 to 247 days, and their progeny (F_1) were fed 25 (1.25 mg/kg/day) and 100 ppm (5 mg/kg/day) amitrole for 141 days. Pups born to parents fed 500 and 1,000 ppm amitrole were small and had atrophic thymuses and spleens indicative of runting; no signs of runting were observed in the 25- and 100-ppm pups. Hyperplasia of the thyroid was observed in all animals fed 25 ppm and higher.

A rat teratology study reported in CDFA (1986) reported no indication of teratogenic effects in offspring when amitrole was given to pregnant rats by gavage on days 6 through 15 of gestation at dose levels of 0, 100, 500, and 1,000 mg/kg/day. A developmental NOEL of 500 mg/kg/day was set based on decreased fetal weight gain at the high dose.

In a rabbit teratology study that CDFA (1986) reviewed, does were administered amitrole by gavage during days 6 through 18 of gestation. Dose levels were 0, 4, 40, and 400 mg/kg/day. Abortions and decreased weight gain of does were observed at 40 mg/kg/day. Increased incidence of structural changes at 40 mg/kg/day resulted in a developmental NOEL of 4 mg/kg/day.

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity Study Results. EPA (1985a) has classified amitrole as a probable human carcinogen (Class B2). Therefore, a cancer risk analysis for amitrole was performed in this risk assessment. Chronic exposure to amitrole through dietary and inhalation routes has resulted in the formation of benign and malignant thyroid tumors in laboratory animals (EPA 1985a).

The following animal studies were reviewed by EPA (1985a) in their *Risk Assessment on Amitrole*.

Rats given 0, 10, 50, and 500 ppm amitrole in the diet for 2 years showed a significant increase in thyroid adenomas (not classified as to type, follicular or interstitial) in the 50-, 100-, and 500-ppm treatment groups when compared to concurrent controls. There was no reported increased incidence of liver tumors in any treatment group. No thyroid function tests were reported. Survival was similar for all groups (Hazelton 1959, as cited in EPA 1985a).

In another study, rats given 0, 1, 10, and 100 ppm amitrole in the diet for 2 years also showed a significant increase in thyroid adenomas and carcinomas (not classified as to type, follicular or interstitial) in the 100-ppm male and female treatment groups when compared to concurrent controls. In addition, a significant increase in pituitary adenomas and carcinomas was observed in the 100-ppm females. The percentage accumulation of radioiodine in the thyroid and thyroid weights were increased in the 100-ppm males and females. No increase in liver tumors was observed, and survivability was similar for all groups (Bayer AG 1979, Steinhoff 1983, both as cited in EPA 1985a).

In another chronic study, rats were pulse fed amitrole in the diet for 2 years in the following manner:

Test Group	Dosing Regimen
A	Control
B	5 ppm (week 1 through 39)/100 ppm (week 40 through 118)
C	1 ppm (week 1 through 39)/20 ppm intermittent[1]
D	3 ppm (week 1 through 39)/60 ppm intermittent[1]
E	10 ppm (week 1 through 39)/100 ppm intermittent[1]

[1]Amitrole diet for 1 month followed by control diet for 1 month, alternating until sacrifice.

A significant increase in thyroid tumors, mainly classified as follicular-type tumors, was observed in male groups B, D, E, and in female groups B and E. In addition, a significant increase in pituitary tumors was observed in the B and E female groups. Thyroid function tests were performed (T_3 and T_4); however, the values were extremely variable and did not correlate with the observed histopathology. No increase in liver tumors was observed, and survivability was similar for all groups (Food and Drug Research Lab 1981, as cited in EPA 1985a).

In a chronic inhalation study, rats were exposed to an unverified amount of amitrole (Food and Drug Research Lab 1981, as cited in EPA 1985a). The incidence of thyroid tumors increased at the unverified dose. Thyroid function tests (T_3 and T_4) were highly variable and did not permit analysis.

Lifetime feeding studies were conducted in hamsters, mice, and rats using 0, 1, 10, and 100 ppm of amitrole (Steinhoff et al. 1983, as cited in EPA 1985a). The results of these studies further confirm the relationship of the disturbance of thyroid function and tumor formation, as well as interspecies variation. Rats showed the most significant changes, as both thyroid and pituitary tumors were observed at 100 ppm. The mouse study showed changes in thyroid organ weights and percent iodine accumulation at 100 ppm; however, no increased incidence in tumor production was observed. EPA considers the thyroid changes seen in mice to be a less profound indicator of thyroid disruption. In the hamster study, neither thyroid function changes nor tumors were observed, thereby indicating that the hamster was the least sensitive species (Steinhoff et al. 1983, as cited in EPA 1985a).

Two additional studies that EPA (1985a) reviewed were reported to have serious experimental design or reporting flaws, but they did indicate amitrole's oncogenic potential in two animal species. Thyroid tumors were reported in mice given 2,192 ppm amitrole for 18 months after the mice were weaned (Innes 1969, as cited in EPA 1985a). Benign and malignant thyroid and liver tumors were also found in rats given 20 and 25 mg/kg/day amitrole in drinking water or 250 and 500 mg/kg/day amitrole in the diet for 10 to 32 months (Napalkov 1969, as cited in EPA 1985a).

In a study conducted by Tsuda et al. (1976) female rats were given 2,500 ppm amitrole in their drinking water for 30 weeks. Weakened

peroxidase activity in follicular cells was followed by the development of goiter. Goiter tissue often proliferated to show malignant adenoma breaking through the capsule, infiltrating into surrounding tissues, and invading blood vessels. An atypical nodular type adenoma was also noted.

As indicated from this rather extensive body of data, amitrole has consistently demonstrated an oncogenic potential in feeding studies using rats, with the thyroid and pituitary as the primary target organs at doses as low as 0.05 ppm (Food and Drug Research Lab 1981, as cited in EPA 1985a). The oncogenic potential in mice is not as clearly demonstrated, as liver and thyroid tumors occurred only after feeding amitrole at doses in excess of 2,000 ppm (Innes 1969, as cited in EPA 1985a). In a comparative species study, doses of 100 ppm amitrole in the diet for 2 years produced an increased incidence of thyroid tumors in rats only, not in mice or hamsters (Steinhoff 1983, as cited in EPA 1985a).

Cancer Potency. Amitrole's cancer potency was estimated by calculating cancer potencies using tumor data from the study by Tsuda et al. (1976) in which rats were given 2,500 ppm in their drinking water and the study by Food and Drug Research Lab (1981, as cited in EPA 1985a) in which rats were alternately fed food with and without amitrole. The higher calculated potency was then used in the calculation of cancer risk.

The data of Tsuda et al. (1976) gave a potency of 0.011 per (mg/kg/day) for all invasive thyroid lesions and 0.00098 per (mg/kg/day) for papillary adenoma. The Food and Drug Research Lab study indicated a cancer potency for thyroid tumors of 0.61 considering only the intermittently dosed groups per (mg/kg/day). The 95-percent upper confidence limit for the potency of 0.61 per (mg/kg/day), based on the Food and Drug Research Lab data, is 1.4 per (mg/kg/day) and is used in this risk assessment.

Mutagenicity. Amitrole did not produce mutagenic effects in several bacterial assays, assays with insects, mammalian in vivo assays, and mammalian sister chromatid exchange assays (USDA 1984) and is not viewed as genotoxic. Positive results were observed in two tests in an invalidated forward mutation system with bacteria (USDA 1984). The chemical also induced in vitro cell transformations in four mammalian cell assays (EPA 1985a). Cell transformation assays are capable of detecting some classes of nongenotoxic carcinogens. EPA (1985a)

concluded and this risk assessment concurs that the extensive genotoxic data base indicates that amitrole is not mutagenic (that is, it does not cause heritable genetic damage) but that amitrole does have oncogenic potential (possibly epigenetic) as demonstrated in the positive in vivo cell transformation studies.

The mutagenic potential of amitrole is summarized in EPA (1985a) as follows:

Amitrole has been evaluated in a variety of mutagenicity test systems. Although positive results were reported by Braun et al., 1977, (using added nitrite) in *Salmonella* and by Venitt and Crofton-Sleigh (1981) in *Salmonella* and *E. coli*, 49 other *Salmonella* gene mutation tests and 9 other *E. coli* tests were negative. The validity of the two positive studies is questionable. The weakly positive results by Carere et al. (1976, 1978, and 1981) were in an invalidated system using unusual bacteria. The mechanisms for these positive results reported for the DNA repair assays cannot be determined without positive gene mutation or chromosome aberration assays. The negative results in the sister chromatid exchange assay in mammalian cells in culture (which is a very sensitive assay) and the chromosome aberration assays in cultured human lymphocytes or in vivo mouse bone marrow cells cast doubt on the significance of the DNA repair assays. Amitrole does not present a potential for heritable genetic effects.

Amitrole is able to induce transformation of cultured cells. It was positive in four in vitro transformation studies using rat and hamster cells (Pienta 1977, Inoue 1981, Dunkel 1981, Styles 1981) following treatment of 0.1 to 100 ug/mL. This test is used to establish the malignant activities of test compounds on mammalian cells in vitro. Cells treated in vitro with chemical carcinogens give rise to foci of cellular growth superimposed on the cell monolayer. If these foci are picked from the cultures, grown to larger numbers, and injected into animals, a malignant tumor will be obtained, in most cases. Therefore, the appearance of piled-up colonies in treated cell cultures is correlated with malignant transformation. In addition, weak cellular transformation capacity was observed in EUE cells (no data presented, only summary) (Benigni 1980).

These transformation assays are not able to determine a mechanism for tumor formation and do not necessarily show that a transformation inducer is genotoxic. These

results support oncogenicity potential but not necessarily mutagenicity potential.

Data Gaps

According to the amitrole registration standard (EPA 1985b), EPA has identified primary skin irritation and dermal sensitization studies as amitrole toxicity data gaps. EPA considers 90-day dermal, primary eye irritation, 90-day inhalation, and teratogenicity study requirements to be only partially satisfied with the current studies. No acute delayed neurotoxicity study exists on amitrole; however, EPA indicates that this study is not required because amitrole "is not structurally related to a known neurotoxin nor does it inhibit cholinesterase" (EPA 1985b).

Amitrole did show transitory skin irritation in the acute dermal toxicity study on rabbits (EPA 1984b). It is assumed in this risk assessment that amitrole may cause skin irritation and dermal sensitization in exposed humans not wearing protective clothing.

The amitrole NOEL for teratogenicity (4 mg/kg/day) is based on the rabbit teratology study reviewed in CDFA (1986).

Atrazine

Toxic Effects Seen in Humans

EPA (1989) established the human reference dose for chronic oral intake at 0.005 mg/kg/day. Schlichter and Beat (1972, as cited in EPA 1987) reported a case of severe contact dermatitis in a 40-year-old farm worker exposed to an atrazine formulation. The clinical signs were red, swollen, and blistered hands with hemorrhagic bullae between the fingers.

Yoder et al. (1973, as cited in EPA 1987) examined chromosomes in lymphocyte cultures taken from agricultural workers exposed to herbicides, including atrazine. The workers showed more chromosomal aberrations during mid-season exposure to herbicides than during the off-season (no spraying). These aberrations included a fourfold increase in chromatid gaps and a 25-fold increase in chromatid breaks. During the off-season, the mean number of gaps and breaks was lower in this group than in controls who were in occupations unlikely to involve herbicide exposure. This observation led the authors to speculate that there is enhanced chromosomal repair during this period of time, resulting in compensatory protection. However, these data may not be representative of the effect of

atrazine, because the exposed workers were also exposed to other herbicides.

Threshold Toxicity In Laboratory Animals

Acute and Subacute Toxicity. Atrazine has a low acute toxicity based on the lowest rat oral LD_{50} of 672 mg/kg (Gaines and Linder 1986, as cited in EPA 1987). EPA (1983) classified atrazine as slightly toxic for acute oral exposure. Acute toxicity symptoms in rats include reduced respiratory rate, motor incoordination, muscle spasms, and hypothermia (Hayes 1982). Dermal exposure to rats did not produce toxicity, and a dermal LD_{50} of greater than 2,000 mg/kg was established (EPA 1983). The dermal LD_{50} in rabbits was 7,550 mg/kg (EPA 1983). Rabbits exposed to technical atrazine failed to show dermal irritation after 24 hours (EPA 1983). The dermal toxicity studies are sufficient to classify the chemical as slightly toxic for dermal effects. An aqueous suspension of technical atrazine has been tested for eye irritation properties in white rabbits. Corneal opacity of minimal severity was present at 1 hour through 72 hours after exposure. Complete reversibility occurred before 7 days. The study is adequate to place the chemical as moderately toxic for eye irritation (EPA 1983).

Acute exposure (1 hour) of rats to atrazine by inhalation revealed that the LC_{50} was greater than a nominal value of 167 mg/L (EPA 1983). The data indicate that atrazine does not possess a high toxicity by way of inhalation. When considered with the oral LD_{50}, EPA concludes that the data are sufficient to classify the chemical as very slightly toxic for inhalation (EPA 1983).

In a subacute study with rats, test animals received 100, 200, 400, or 600 mg/kg atrazine for 14 days. Renal effects observed included increased elimination of sodium, potassium, and chloride, decreased levels of creatinine clearance, increased urine protein levels, and increased lactate dehydrogenase activity. These results suggest that the nephrotoxic properties of atrazine may affect not only its excretion but also increase its toxicity in the kidney (Santa Maria et al. 1986).

Subchronic and Chronic Toxicity. In a 2-year feeding study in which beagles were fed up to 1,500 ppm (37.5 mg/kg/day) of the 80W formulation, body weights were lowered at the HDT but not at the mid-dose level. Reduced food intake, increased adrenal weights, occasional tremors and stiffness in the limbs, and reduced hematocrit values were also

noted at the high dose. Liver and heart weights were increased, and food intake was reduced in the mid-dose females. The systemic NOEL for this study was reported as less than 15 ppm (0.38 mg/kg/day) (EPA 1988) and is the systemic NOEL used in this risk assessment.

A 2-year feeding study with rats resulted in a systemic NOEL of 70 ppm (3.5 mg/kg/day), based on reduced weight gains, irritability in males, and pale appearance and probable anemia in females at the next higher dose (CDFA 1986). A 22-month chronic feeding/oncogenicity study in mice resulted in a systemic NOEL of 300 ppm (36 mg/kg/day) with decreased male and female body weights and increased cardiac thrombi (blood clots) at the lowest effect level of 1,500 ppm (180 mg/kg/day) (EPA 1988).

Reproductive and Developmental Toxicity. In a rat teratology study, atrazine was given at dose levels of 0, 10, 70, and 700 mg/kg/day during days 6 to 15 of gestation (Ciba-Geigy 1984a, as cited in EPA 1987). Excessive maternal mortality was observed at 700 mg/kg/day, but none occurred at lower doses. Reduced weight gain and food consumption were noted at 70 and 700 mg/kg/day. Fetal weights were severely reduced at 700 mg/kg/day; delays in skeletal development occurred at 70 mg/kg/day; and dose-related runting was noted at 10 mg/kg/day and above. The maternal NOEL was 10 mg/kg/day, and the fetotoxic NOEL was less than 10 mg/kg/day (LDT).

In a second teratology study with rats, treatment at 100, 500, and 1,000 mg/kg on days 6 to 15 of gestation caused an increase in embryonic and fetal deaths at 500 mg/kg. The two higher doses also decreased the mean weights of fetuses and retarded skeletal development. A NOEL for maternal toxicity and fetotoxicity (embryonic resorption) was reported as 100 mg/kg. Teratogenic effects were not observed at any dosage up to and including 1,000 mg/kg (HDT) (Ciba-Geigy 1971, as cited in EPA 1987).

In a rabbit teratology study reported by Ciba-Geigy, dose levels of 0, 1, 5, or 75 mg/kg/day were given by gavage during gestation days 7 through 19. Decreased body weight gain and food consumption occurred in does in the mid- and high-dose groups. At 75 mg/kg/day, increased resorption rate, reduced fetal weights, and delays in ossification were observed. No teratogenic effects were indicated. The NOEL was established as 1

mg/kg/day for maternal toxicity in this study (Ciba-Geigy 1984a, as cited in EPA 1988).

A two-generation reproduction study in rats resulted in a NOEL of 10 ppm (0.5 mg/kg/day), based on decreased pup weights at the lowest effect level of 50 ppm (2.5 mg/kg/day) (EPA 1988). This is the reproductive NOEL used in this risk assessment. A three-generation reproduction study in rats did not result in any adverse effects at the highest dose tested of 100 ppm (5 mg/kg/day) (EPA 1988).

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity Study Results. Available data suggest that atrazine may be carcinogenic; therefore, a cancer risk analysis was done for atrazine in this risk assessment. In a 2-year feeding/oncogenicity study, rats were fed technical atrazine at doses of 0, 10, 70, 500, and 1,000 ppm in the diet (EPA 1988). Dose-related increases in adenocarcinomas and fibroadenomas were observed in female mammary glands. Results were statistically significant at 70 ppm (3.5 mg/kg/day) and above for carcinomas and at 1,000 ppm (50 mg/kg/day) and above for adenomas and fibroadenomas. No oncogenic effects were observed in males. A 22-month chronic feeding/oncogenicity study revealed no oncogenic findings in mice (EPA 1988).

An 18-month mouse feeding study showed no tumor induction when mice were given 21.5 mg/kg by gavage from days 7 to 28 of age, then given 82 ppm (12.3 mg/kg/day) for the remainder of the experiment (Innes et al. 1969, as cited in USDA 1984).

Cancer Potency. Based on the information available, EPA (1989) has classified atrazine as a possible human carcinogen (Group C). Atrazine cancer potency for this risk assessment was calculated based on the rate of mammary tumor formation in female rats in the 2-year chronic feeding oncogenicity study reported by CDFA (1986). The cancer potency in rats, estimated using the one-hit cancer model, is 0.03 per (mg/kg/day) (USDA 1986). The cancer potency adjusted for humans is 0.22 per (mg/kg/day) (EPA 1986).

Mutagenicity. Atrazine did not induce mutations in EPA-validated microbial assay systems, which included a recombination and conversion assay in strains of *B. Subtilis, E. coli,* and *S. typhimurium;* an Ames assay with and without metabolic activation in four strains of bacteria; and a DNA repair assay using rat hepatocyte (EPA 1988).

Atrazine was reported to induce sex-linked recessive lethal mutations in the fruit fly (*Drosophila melanogaster*) in one of two studies (USDA 1984).

In vivo studies measuring chromosome aberrations in rodent bone marrow and dominant lethal mutations in mice were reported positive at dose levels of 2,000 and 1,500 mg/kg, respectively (USDA 1984).

As discussed earlier, Yoder et al. (1973, as cited in EPA 1987) showed that there were more chromosomal aberrations in lymphocyte cultures taken from agricultural workers during mid-season exposure to herbicides, including atrazine, than during the off-season (no spraying).

Atrazine was positive for mutagenicity in eight gene mutation studies and negative in nine others. Three of these positive responses were in tests with the fruit fly that measured gene mutations in germ cells. Positive results were also obtained in tests with mice that measured chromosome alterations in germ cells. Positive responses in these types of assays indicate a potential for mutagenic hazard. Chromosome aberrations in bone marrow cells in vivo support this conclusion. However, these in vivo responses were observed only at very high levels of atrazine equal to or exceeding 1,500 mg/kg (USDA 1984).

Atrazine is not genotoxic in bacteria or yeast directly or with the typical rodent S9 activation. Yeast tests for mitotic crossing over and mutation do show positive responses but only when tested with atrazine incubated with plant cell extracts. Bacteriophage T4 mutation and a *B. subtilis* test for repairable DNA damage were negative with atrazine alone. A *Streptomyces coelicolor* mutation assay was positive, but this assay appears to have little reliability to discriminate true positives and false positives (USDA 1984).

In vitro tests with mammalian cells also fail to respond to atrazine directly or with mammalian metabolism (aberrations and sister chromatid exchange tests in hamster cells were negative), but when hamster V79 cells were exposed to atrazine in the presence of plant cell extracts, the chemical was reported to be mutagenic. Positive unscheduled DNA synthesis (UDS) effects in EUE cells were also reported with atrazine plus plant cell extracts (USDA 1984).

Atrazine was reported to be genotoxic directly in plants (mutations at the waxy locus in corn

and chromosome aberrations in plant cells) and in the mold *Aspergillus nidulans* (crossing over). Mutation studies in *Aspergillus* were conducted with plant cell extracts. In the presence of plant cell extracts, a mutation to 8 azaguanine resistance was reported for *Aspergillus* (USDA 1984).

Atrazine represents an unusual situation because many of the positive genotoxicity studies were conducted using a metabolic activation system of plant origin. Atrazine alone or when tested in vitro in tests using animal metabolic systems resulted in variable responses. Because plant metabolism is not generally considered important in developing a human hazard assessment, the relevance of the "plant-activated" tests in a risk analysis is doubtful.

Although all EPA-validated mutagenicity assays are negative, many studies in the open literature indicate a possible human germ cell mutagen. For the purpose of this analysis, it is conservatively assumed that atrazine is a possible human germ cell mutagen at high levels of exposure. The degree of hazard to humans from low levels of exposure is likely to be minimal.

N-Nitrosoatrazine

N-nitroso derivatives of some herbicides are carcinogenic and mutagenic (Young and Khan 1978, Braun et al. 1977). Little information is available on the formation of these compounds under normal conditions of herbicide application or on their metabolism in soil and water (Greenhalgh 1978). Concerns have been raised over the potential for the nitrosation of atrazine to N-nitrosoatrazine (NNA) under field conditions and the potential toxicity of this compound. No information is available on the toxicity, mutagenicity, or carcinogenicity of N-nitrosoatrazine.

Kearney et al. (1977) have examined the formation and degradation of NNA in soils and aquatic environments. Their results indicate that the formation of NNA is highly unlikely under normal application rates of atrazine (2 ppm) in agricultural soils of pH 5 to 7. Kearney et al. used elevated levels of nitrogen fertilizer (approximately 100 ppm), and these rates are much higher than those used in forestry. In an examination of its persistence, most of the NNA added to soil was converted relatively quickly to atrazine by denitrosation.

The degradation of NNA in aquatic environments is very rapid primarily because of photolysis. However, the formation of NNA in

ground water contaminated with atrazine is unknown (Wolfe et al. 1976).

Data Gaps

According to EPA (1989), there are no data gaps for atrazine.

Bromacil

Toxic Effects Seen in Humans

The human reference dose for chronic oral intake of bromacil was established by EPA at 0.13 mg/kg/day, based on 2-year feeding studies in rats with NOEL of 12.5 mg/kg/day (EPA 1989). A safety factor of 100 was applied for inter- and interspecies variation.

Threshold Toxicity in Laboratory Animals

Acute and Subacute Toxicity. Based on the lowest acute oral LD_{50} of 3,998 mg/kg in rats, bromacil can be classified as slightly toxic. The acute dermal LD_{50} for rabbits is 2,000 mg/kg, and the acute inhalation LC_{50} in rats is greater than 57.6 mg/L. Bromacil is a mild eye irritant and is very slightly irritating to rabbits' skin (EPA 1986).

Subchronic and Chronic Toxicity. In a 90-day feeding study using an 80-percent wettable powder formulation, rats were given bromacil doses of 0, 50, 500, or 2,500 ppm. The high dose was raised to 5,000 ppm the sixth week. At 5,000 ppm, lower growth rates, decreased erythrocyte count, increased thyroid activity, and enlargement of centrolobular cells of the liver were observed. The NOEL for this study was 500 ppm (25 mg/kg/day). In a 2-week feeding study, rats exhibited gastrointestinal disturbance and CNS incoordination after receiving 10 doses of 1,035 mg/kg each (EPA 1986).

In a 2-year dog feeding study, beagles were given 0, 50, 250, or 1,250 ppm bromacil (82 to 83.4 percent) in the diet. At 1,250 ppm, some decline in body weights was observed. The NOEL was 250 ppm (6.25 mg/kg/day) (EPA 1986, CDFA 1986).

In a 2-year rat feeding/oncogenicity study, dose levels were 0, 50, 250, and 1,250 ppm. Weight retardation was observed at the 1,250 ppm level. Thus, the NOEL for this study is 250 ppm (12.5 mg/kg/day) (EPA 1986).

In a 2-year mouse feeding/oncogenicity study, mice were given 0, 250, 1,250, or 5,000 ppm. At the lowest dose, testicular abnormalities in the form of focal atrophy of seminiferous

tubules were observed. In addition, carcinomas and hepatocellular adenomas were observed in males at all dosage levels (EPA 1988).

Reproductive and Developmental Toxicity. In rat and rabbit teratology studies, no teratogenic, fetotoxic, or maternal toxic effects were observed at the highest dose tested (165 mg/m^3 converted to 7.92 mg/kg in a rat inhalation study, and 250 ppm converted to 7.5 mg/kg in a rabbit dietary study). No reproductive effects were observed in a three-generation rat reproduction study at 250 ppm (12.5 mg/kg/day)—the only dose tested (EPA 1986).

Nonthreshold Effects In Laboratory Animals

Carcinogenicity. Based on positive results in a mouse oncogenicity study, a cancer risk analysis was done for bromacil in this risk assessment. In a 2-year feeding/oncogenicity study in rats, no oncogenic effects were observed at dietary levels of up to 1,250 ppm (HDT) (EPA 1986). The 2-year mouse feeding/oncogenicity study, discussed previously, showed an increased incidence of hepatocellular adenomas and carcinomas at the 5,000-ppm (750 mg/kg/day) dose level (EPA 1988a). Bromacil is classified as a possible human carcinogen (Group C) based on the available data (EPA 1987).

Cancer Potency. Bromacil cancer potency for this risk assessment was based on the rate of liver tumor formation in male mice in the 2-year feeding study. The estimated cancer potency is 0.0038 per (mg/kg/day) (EPA 1985).

Mutagenicity. Because of the potent mutagenicity of 5-bromouracil, which is a structurally related chemical, the metabolic fate of bromacil has been examined to determine whether the formation of 5-bromouracil occurs in vivo. Metabolic fate studies indicate that 5-bromouracil was not isolated from the urine and feces of rats exposed to bromacil or from the urine of bromacil production plant workers (DOE 1983).

Bromacil showed negative results in microbial assays for gene mutation, a mammalian in vivo assay, a mouse dominant lethal assay, and mammalian and microbial assays for DNA damage (EPA 1987). In one Ames assay, bromacil did induce reverse mutation (EPA 1987). Bromacil caused weakly positive results in one *Drosophila* recessive lethal assay and negative results in another (EPA 1987). Thus, the weight of evidence reviewed

for this risk assessment indicates that bromacil presents a low risk of mutagenicity.

Data Gaps

According to EPA (1989), there are no existing data gaps for bromacil.

Chlorsulfuron

Toxic Effects Seen In Humans

EPA established a reference dose (RfD) of 0.05 mg/kg/day for chronic oral exposure to chlorsulfuron based on a 2-year oral toxicity study in rats with a NOEL of 5 mg/kg/day (EPA 1989). An uncertainty factor of 100 was applied to the NOEL to derive the RfD.

Threshold Toxicity In Laboratory Animals

Acute and Subacute Toxicity. Based on acute oral LD$_{50}$ values of 5,545 mg/kg and 6,293 mg/kg for male and female rats, respectively (Du Pont 1982), chlorsulfuron is classified as very slightly toxic. The acute dermal LD$_{50}$ for rabbits was found to be greater than 3,400 mg/kg (Du Pont 1982), and the primary dermal irritation study in rabbits showed that chlorsulfuron was not a primary irritant (EPA 1988b). The acute inhalation LC$_{50}$ for rats was found to be greater than 5.9 mg/L/4 hours (EPA 1988b). In a primary eye irritation study in rabbits, instillation of 10 mg of chlorsulfuron into eyes of rabbits induced very mild temporary conjunctival irritation (Du Pont 1982).

Subchronic and Chronic Toxicity. Subchronic oral toxicity tests with chlorsulfuron were carried out in rats, mice, and dogs. A 90-day feeding study in rats established a systemic NOEL of 100 ppm (5 mg/kg/day) for chlorsulfuron based on slight decrease in plasma creatinine and slight increase in hematocrit in the 500-ppm group (EPA 1988b). A 90-day feeding study in mice established a systemic NOEL of 2,500 ppm (365 mg/kg/day) for chlorsulfuron based on decreases in erythrocyte counts and relative liver weights and increases in corpuscular volume and mean corpuscular hemoglobin in the 5,000-ppm group (EPA 1988b).

A 180-day feeding study in dogs established a NOEL of greater than 2,500 ppm (62.5 mg/kg/day), the highest dose tested for chlorsulfuron (EPA 1988b).

Chronic oral toxicity tests with chlorsulfuron were carried out in rats and mice. Groups of rats were fed 0, 100, 500, or 2,500 ppm

chlorsulfuron in the diet for 2 years (EPA 1988b). Rats in the 500-ppm group showed reduced body weight gains. Based on the adverse effect in these rats, EPA established a systemic NOEL of 100 ppm (5 mg/kg/day) for chlorsulfuron (EPA 1988b). In a 2-year feeding study, groups of mice were fed 0, 100, 500 or 5,000 ppm chlorsulfuron in the diet (EPA 1988b). The mice in the 5,000-ppm group showed reduced body weight gains and food consumption. Based on the adverse effects in these mice, EPA established a systemic NOEL of 500 ppm (75 mg/kg/day) for chlorsulfuron (EPA 1988b).

Reproductive and Developmental Toxicity. In a three-generation reproduction study, groups of rats were fed 0, 100, 500, or 2,500-ppm chlorsulfuron for 103 days before the reproduction study began. This study resulted in a maternal NOEL of 500 ppm (25 mg/kg/day), based on reduction in mean body weight gains in the 2,500-ppm group. A reproductive NOEL of 500 ppm (25 mg/kg/day) was established, based on reduction in fertility indices at 2,500 ppm (125 mg/kg/day) (EPA 1988b).

Teratogenicity tests with chlorsulfuron were carried out in rats and rabbits. A rat teratogenicity study established a teratogenic, maternal, and fetotoxic NOEL of greater than 2,500 ppm (125 mg/kg/day), the highest dose tested (EPA 1988b). In a rabbit teratogenicity study, a teratogenic NOEL of greater than 75 mg/kg/day (the highest dose tested) was reported, and a fetotoxic NOEL of 25 mg/kg/day was reported, based on increased incidence of resorption in the 75-mg/kg/day group (EPA 1988).

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity. Available data do not indicate that chlorsulfuron is carcinogenic. In 2-year rat and mouse carcinogenicity studies, no carcinogenic effects were observed at dosage levels up to 2,500 ppm (125 mg/kg/day) and 5,000 ppm (750 mg/kg/day), the highest doses tested in rats and mice, respectively (EPA 1988). According to EPA's Integrated Risk Information System (EPA 1988a), this chemical has not been evaluated for evidence of human carcinogenic potential.

Mutagenicity. Chlorsulfuron was nonmutagenic in an Ames bacterial assay, a Chinese hamster ovary mammalian cell assay, a rat dominant lethal assay, an in vitro cytogenetic assay, and a DNA repair assay (du Pont 1982). Based on these results, chlorsulfuron

is not considered to be a potential human mutagen in this risk assessment.

Data Gaps

EPA's Integrated Risk Information System (EPA 1988a) and Reference Dose Tracking Report (EPA 1989) reported that there are no data gaps for chlorsulfuron.

Clopyralid

Toxic Effects Seen in Humans

No human toxicity data on clopyralid were available in current literature. EPA's Office of Pesticide Programs established a reference dose of 0.5 mg/kg/day, based a on 2-year rat feeding study with a NOEL of 50 mg/kg/day (1989).

Threshold Toxicity in Laboratory Animals

Acute and Subacute Toxicity. Based on acute oral LD_{50} values for male rats, which range from 4,300 to greater than 5,000 mg/kg (WSSA 1983), clopyralid is classified as slightly to very slightly toxic. The acute inhalation LC_{50} for rats was found to be greater than 5.03 mg/L/1 hour, and the dermal LD_{50} for rabbits was found to be greater than 2,000 mg/kg (WSSA 1983). A 14-day dermal irritation study in rabbits showed that clopyralid caused slight or questionable irritation at the unspecified dose levels (EPA 1988). In an eye irritation study, however, clopyralid was a severe irritant in rabbits (Agrochemicals Handbook 1989).

Subchronic and Chronic Toxicity. A 90-day rat feeding study with clopyralid resulted in a systemic NOEL of greater than 150 mg/kg/day (EPA 1988). A 1-year dog feeding study with clopyralid resulted in a systemic NOEL of 100 mg/kg/day (EPA 1988).

Three chronic feeding tests with clopyralid were carried out in rats and mice. In the first study, groups of rats were fed clopyralid at dose levels of 0, 5, 15, 50 or 150 mg/kg/day for 2 years (EPA 1988). The female rats in the 150/mg/kg/day group showed a reduction in mean body weight. Based on this adverse effect in the female rats, (EPA established) a systemic NOEL of 50 mg/kg/day for clopyralid. However, EPA (1988) rated this study "supplementary."

In the second study, groups of rats were fed clopyralid at dose levels of 0, 15, 150, or 1,500 mg/kg/day for 2 years (EPA 1988). The rats in the 150-mg/kg group showed hyperplasia and thickening of the limiting ridge

of the stomach. Based on these adverse effects, (EPA established) a systemic NOEL of 15 mg/kg/day for clopyralid. However, EPA (1988) also rated this study "supplementary."

In the third study, groups of mice were fed clopyralid at dose levels of 0, 100, 500, or 2,000 mg/kg/day (EPA 1988) for 2 years. EPA established a systemic NOEL of 500 mg/kg/day for clopyralid, based on decreased body weights in male mice in the 2,000-mg/kg/day group. A "minimum" rating was given to this study (EPA 1988).

Reproductive and Developmental Toxicity. In a two-generation rat reproduction study in which rats were fed clopyralid at dose levels of 0, 150, 500, or 1,500 mg/kg/day, a reproductive NOEL was established to be greater than 1,500 mg/kg/day (EPA 1988). This study resulted in a systemic NOEL of 500 mg/kg/day for clopyralid, based on decreased terminal body weights in the 1,500-mg/kg/day group.

Teratogenicity studies with clopyralid were carried out in rabbits and rats (EPA 1988). A rabbit teratogenicity study established a teratogenic, maternal, and fetotoxic NOEL of greater than 250 mg/kg/day, the highest dose tested. In a rat teratogenicity study, teratogenic and fetotoxic NOELs of greater than 250 mg/kg/day were reported, and a maternal NOEL of 75 mg/kg/day was reported based on decreased body weight gains and food consumption in the 250-mg/kg/day group, the highest dose tested.

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity Studies. Available data do not indicate that clopyralid is carcinogenic. In a mouse 2-year dietary feeding/carcinogenicity study, carcinogenic effects were not found at dose levels up to 2,000 mg/kg/day (EPA 1988). Two-year feeding/carcinogenicity tests with clopyralid in rats were reported in two studies (EPA 1988). In both studies, carcinogenic effects of clopyralid were not found at the highest dose levels of 150 or 1,500 mg/kg/day.

Mutagenicity. Clopyralid was nonmutagenic in the following four assays reported by EPA (1988) in the tox one-liner on clopyralid: a dominant lethal assay, an in vivo cytogenetic assay, an Ames bacterial assay, and a host mediated assay.

Data Gaps

Based on the EPA (1988) tox one-liner for clopyralid, there appears to be no acceptable data for primary DNA damage. Although an unscheduled DNA synthesis assay was submitted to EPA, it was deemed unacceptable. According to EPA (1989), no other data gaps exist for clopyralid.

2,4-D

Toxic Effects Seen in Humans

The human reference dose for chronic oral exposure to 2,4-D was established at 0.01 mg/kg/day by EPA (1989), based on a 2-year rat oral toxicity study with a NOEL of 3.0 mg/kg/day. An uncertainty factor of 100 was applied in the calculation of the human reference dose to account for inter- and interspecies variation. The World Health Organization established a human reference dose at 0.3 mg/kg/day (EPA 1989).

Peripheral neuropathy has been reported to result in humans from dermal exposure to 2,4-D. In one study, Goldstein et al. (1959) reported three cases in agricultural workers following dermal exposure to 2,4-D. The neuropathy was characterized by progressive numbness, aching of the extremities, muscular fasciculation, enervation of muscles, and decreased conduction velocity in the ulnar nerve. The condition may be partially or totally reversible, depending on the dose level and the individual exposed (Todd 1962, Berkeley and Magee 1963, and Wallis et al. 1970). In one patient, only partial recovery was reported, even after 3 years of treatment (Goldstein et al. 1959). His estimated exposure was 60 cubic centimeters of a 10-percent ester solution, approximately 60 mg/kg.

Several epidemiological investigations have been conducted to examine the link between human phenoxyacid herbicide exposure and cancer. In the mid and late 1970s, Hardell and colleagues (Hardell and Sandstrom 1979, Eriksson et al. 1981, Hardell et al. 1981) conducted a series of case-control studies in rural Sweden. These studies found a significant increase of fivefold to sixfold in the relative risk of soft-tissue carcinomas, Hodgkin's disease, and non-Hodgkin's lymphoma among farmers using various herbicides. However, because of selection bias, observation bias, and uncontrolled confounding variables, many experts have questioned the validity of the results of these studies (Colton 1986).

In a Danish cohort study of workers manufacturing phenoxy herbicides, a significant increase in risk of soft-tissue sarcoma (STS) was found, but no similar increase in malignant lymphoma (Lynge 1985). The cancer risk among persons manufacturing and packaging phenoxy herbicides was equivalent to the cancer risk in the Danish population (Lynge 1985). A recent Swedish cohort study found no significantly increased relative risk (0.9) of STS in Swedish agricultural and forestry workers exposed to phenoxy acid herbicides (Wiklund and Holm 1986). In addition, a case-control study conducted in New Zealand by Smith et al. (1984) was negative for soft-tissue carcinomas, showing an estimated relative risk of 1.3.

Recently, Hoar et al. (1986) completed a case-control study in Kansas examining the risk of lymphoma and STS in men from agricultural herbicide exposure. The study found no association between exposure and soft-tissue carcinoma or Hodgkin's disease, but a significant association was observed for non-Hodgkin's lymphoma (NHL) and phenoxyacetic acid herbicide exposure, especially 2,4-dichlorophenoxyacetic acid exposure. In addition, individuals exposed to herbicides for more than 20 days per year had a sixfold increase in NHL. Nonetheless, this study suffers from the same inherent limitations as other case-control studies, mainly that it relies on the recall ability of the subject or their next of kin to determine their exposure status. If their recall is faulty, misclassification occurs. It is especially difficult to assess exposure-disease relationships in these types of epidemiological studies (NRC 1986). For example, it is possible to have common exposures to other carcinogenic agents or other factors that result in disease but are not discovered in the interview and confound the results. Thus, uncontrolled confounding factors in observational epidemiological studies can be particularly troublesome in interpreting the results. However, the apparent dose-response relationship observed in the Hoar et al. (1986) study for NHL is of public health concern and needs further examination. It should be noted that at least two additional studies are now under way that should be helpful in assessing risk to humans from the use of 2,4-D and other phenoxy herbicides (Colton 1986).

In a recent review of the Hoar et al. (1986) study conducted for EPA, Brian MacMahon, M.D., Ph.D., of the Harvard School of Public Health, concluded:

> In my opinion the weight of evidence does not support the conclusion that there is an

association between exposure to 2,4-D and NHL. It is axiomatic that, except when relative risks are very high—and sometimes even then—no single study will establish an association between an exposure and an outcome. The acceptance of an association depends on a number of studies showing consistent results across populations and across different epidemiologic methods. The study of Hoar et al. is a strong study—strong enough on its own to establish a hypothesis of relationship of exposure to 2,4-D with some small proportion of cases of NHL—a hypothesis that clearly deserves attempts at refutation or support in other populations. When one attempts to place the results of this study among the results of those published previously, the picture becomes very confusing—much more than if Hoar et al. had been the only study published. Taken as a whole, I believe that the weight of evidence indicates that an association between 2,4-D and NHL remains a hypothesis that is still to be tested. I am unwilling to speculate as to whether 2,4-D causes NHL (or some cases of NHL) until the evidence is clear that there is an association between them (MacMahon 1986).

Also, in a cohort study of forestry workers in Ontario, no evidence of increased mortality risk or cancer risk was observed in forestry workers after 15 or more years of employment associated with phenoxy herbicide use (Green 1986). The forestry workers had been employed by Ontario Hydro from 1950 through 1982.

The Canadian Centre for Toxicology (1987) has reviewed recent case-control studies of phenoxy herbicides. A study conducted in western Washington State reported no overall increased risk associated with past occupational exposure to phenoxy herbicides for STS or NHL (Woods et al. 1987, as cited in Canadian Centre for Toxicology 1987). There was an elevated risk of NHL for men who had been farmers, forestry herbicide applicators, and those potentially exposed to phenoxy herbicides for 15 years or more during the 15-year period before cancer diagnosis. However, exposure to 2,4-D was not singled out.

Another study reviewed by the Canadian Centre for Toxicology (1987) is being conducted by the National Cancer Institute in Iowa and Minnesota. Preliminary results indicate no overall increased risk for NHL associated with living or working on a farm and a slightly elevated (but not significant) risk in persons using 2,4-D (Cantor and Blair 1986,

as cited in Canadian Centre for Toxicology 1987). The investigators have decided to recontact subjects to gather more information on the number of days per year of pesticide use.

After reviewing the 2,4-D epidemiology studies, the Canadian Centre for Toxicology (1987) concluded that there is limited evidence of carcinogenicity in humans from exposure to phenoxy herbicides, and there is inadequate evidence to classify 2,4-D as a carcinogen.

Threshold Toxicity in Laboratory Animals

Acute and Subacute Toxicity. 2,4-D can be classified as moderately toxic in rats with an LD_{50} of 375 mg/kg (EPA 1986a). The acute dermal LD_{50} of 2,4-D (21.1 percent active ingredient) in rabbits is greater than 3,980 mg/kg (EPA 1986a). Skin absorption of 2,4-D is limited. Feldmann and Maibach (1974) found that approximately 5 to 6 percent of the 2,4-D dermally applied to humans was recovered in the urine. When dermal contact continues, nausea, vomiting, muscular weakness, and diarrhea have been reported (Newton and Dost 1984). 2,4-D is a severe eye irritant (EPA 1986a), and acute eye irritation can result from occupational exposures (WHO 1984). 2,4-D ingestion or skin exposure in humans can cause irritation to the gastrointestinal tract, chest pain, and muscle twitching. Ingestion of large doses of 2,4-D causes gastroenteritis, skeletal and cardiac myotonia, and central nervous system depression in humans. A human dose of 80 mg/kg of the diethylamine salt of 2,4-D caused congestion of all organs, degenerative nerve cells, and death. Accidental swallowing of 110 mg/kg of isooctyl ester of 2,4-D caused muscle twitching and paralysis, although the individual recovered in 24 hours (Mullison 1981, as cited in USDA 1984).

Peripheral neuropathy has not been seen in laboratory animals dermally exposed to 2,4-D. Four groups of male and female Fischer CDF 344 rats (15 rats per group) were used in a study to determine whether repeated dermal exposure of rats to 2,4-D would result in pharmacological or toxicological effects on the peripheral nervous system. The skin of the animals in the three treatment groups was painted with a 12-percent 2,4-D amine solution for 2 hours per day, 5 days per week, for 3 weeks. Control animals were treated with tap water. Dermal exposure to 2,4-D resulted in two systemic effects: (1) treated rats weighed less than control rats, and (2) the kidneys of treated rats weighed more than those of the control rats. Even though the rats had clear

systemic effects of exposure to 2,4-D, there were no treatment-related changes in the function or structure of the nervous system (EPA 1986b).

Subchronic and Chronic Toxicity. In a 90-day rat subchronic feeding study, histopathological abnormalities were observed at the lowest dose tested of 1.0 mg/kg/day (1986a).

EPA (1985) has reviewed results from the first year of a chronic feeding study on rats. Based on renal effects, a NOEL of 1 mg/kg/day was established; the lowest effect level was 5 mg/kg/day. Based on this study and using a hundredfold safety factor, EPA has established a provisional ADI of 0.01 mg/kg/day (1989).

Reproductive and Developmental Toxicity. Schwetz et al. (1971) examined the effects of 2,4-D and two esters of 2,4-D on fetal development and neonatal growth and survival in rats. Dose levels up to the maximum tolerated dose of 87.5 mg/kg/day were administered to the laboratory animals on days 6 through 15 of gestation. The fetuses then were delivered by cesarean section on day 20 of gestation and examined for anomalies. The anomalies observed include decreased fetal body weight, subcutaneous edema, delayed bone ossification, lumbar ribs, and wavy ribs. Since none of these anomalies interferes with fetal or neonatal development and survival, they were classified in this study as neither embryotoxic nor fetotoxic. No treatment-related teratogenic responses were observed. At the highest dose level, decreased viability and lactation indices were observed. Therefore, a reproductive NOEL of 25 mg/kg/day was established.

EPA has recently reviewed a rat teratology study that used an acid form of 2,4-D (EPA 1985). Based on fetotoxicity and delayed ossification, a NOEL of 25 mg/kg/day was established; the lowest effect level was found to be 75 mg/kg/day.

A recent multigeneration rat study was conducted at dose levels of 0, 5, 20, and 80 mg/kg/day. During gestation and lactation of the original parents, the female high-dose group was actually receiving about 120 mg/kg/day. Adverse effects on the original parents in this dose group and their offspring were excessive, and the 80-mg/kg/day dosage level was terminated (Mullison 1986). According to EPA (1986b), the results showed no effects at 5 mg/kg/day. At the next higher dose tested (20 mg/kg/day),

however, maternal body weights and pup weights decreased (EPA 1986b).

The n-butylester of 2,4-D was analyzed for immunotoxity in an acute and subacute oral study, an acute and subacute dermal study, and a teratology study with mice. In the acute dermal study, mice exhibited suppressed antibody production against sheep red blood cells at high exposure levels (500 mg/kg). This response was believed to be a secondary manifestation of clinical signs observed rather than a direct immunological effect. In the subacute dermal study, antibody production was not suppressed, but it did enhance lymphocyte proliferative responses. The authors concluded that the results of this study suggest that 2,4-D esters are unlikely to have any major immunotoxicological significance (Blakley and Schiefer 1986).

In the acute and subacute oral studies, immunostimulatory effects were observed at relatively high exposures to 2,4-D (100 to 200 mg/kg/day). It was also concluded that these immune alterations would not have any major toxicological significance (Blakley 1986). Likewise in the teratology study, no net suppressive effect was observed, and although subtle effects were noted in lymphocyte blastogenesis, the authors concluded that the 2,4-D ester was unlikely to be of any immunotoxicological or teratological significance (Blakley and Blakley 1986).

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity. There is much controversy and little definitive evidence from laboratory studies and epidemiology studies to indicate that 2,4-D may be carcinogenic. Nevertheless, a cancer risk analysis was done for 2,4-D in this risk assessment. Several chronic toxicity/oncogenicity studies have been reported in the literature using various esters of 2,4-D. Innes et al. (1969) reported that the maximum tolerated dose (46.4 mg/kg/day) of butyl, isopropyl, or isooctyl esters of 2,4-D was fed to two strains of mice for up to 78 weeks with no significant increase in the tumor incidence observed at a 95-percent confidence level.

A study was reported by Hansen et al. (1971) in which, over a period of more than 2 years, rats were fed 2,4-D at 0, 5, 25, 125, 625, and 1,250 ppm, and dogs were fed 2,4-D at 0, 10, 50, 100, and 500 ppm. In the dogs, no increased tumor incidence was observed, and no other lesions were attributed to 2,4-D. The rats showed a high incidence of tumors (30 percent) in both the treated and untreated

(control) groups. The male rats had a significantly higher incidence of malignant tumors in the high-dose group (1,250 ppm), and the female rats showed a trend toward increased tumor formation with the logarithm of dose. However, Hansen et al. (1971) concluded that, because the tumors were not target organ types but were randomly distributed types normally found in aging Osborne-Mendel rats and because survival rates were not affected, the data "support the pathological interpretation that a carcinogenic effect of 2,4-D has not been shown." However, one expert, Dr. M. Reuber, has reexamined the data and challenged the conclusion that no carcinogenic effect was demonstrated (Halts 1980, as cited in USDA 1984).

In a study of adult female sheep that were examined at slaughter, exposure to phenoxy herbicides was associated with significant increases in the rate of small intestinal adenocarcinoma (Newell et al. 1984). Tumor rates rose significantly with the total number of phenoxy sprays used on the farm.

According to the World Health Organization (WHO) (1984), "the carcinogenic potential of 2,4-D and its derivatives such as the amine salts and esters has not been adequately tested. The reports on animal bioassay carried out so far are either too brief for proper evaluation or have been the subject of scientific controversy."

EPA's (1986c) review of a long-term study on the oncogenic potential of 2,4-D reported an increased incidence of brain tumors in rats. EPA has requested an independent expert to review the brain tissue slides from this study, and may request a Scientific Advisory Panel review. Therefore, EPA does not believe it is appropriate at this time to derive a specific numerical estimate of cancer potency based on the new data. However, EPA has stated that, based on their preliminary review, the level of cancer potency indicated by the reported results would be of about the same order of magnitude as the potency value based on the Hansen study (EPA 1986c).

At 106 weeks, a preliminary pathology report from a recent mouse study found that 2,4-D was not oncogenic at dosages of 1, 15, and 45 mg/kg/day (Hazelton Laboratories 1986).

Cancer Potency. Because of the uncertainty about the carcinogenicity of 2,4-D, a cancer risk analysis was conducted for 2,4-D in this risk assessment. 2,4-D cancer potency was calculated based on the rate of tumor

formation in the female Osborne-Mendel rats studied by Hansen et al. (1971). This is the species and sex that has exhibited the highest increase in tumor formation after 2,4-D administration. All tumors were considered, although many of them were benign. The 95-percent upper confidence limit of the cancer potency, calculated by Crump (1983) using the GLOBAL 82 computer program, was 0.00503 per (mg/kg/day). In this risk assessment an interspecies extrapolation factor of 5.8 was used to adjust the rat cancer potency for humans (K=(human weight/test animal weight) 1/3; average human weight = 70 kg, average rat weight = 350g). The adjusted potency is 0.029 per (mg/kg/day). A preliminary review of an additional long-term oncogenicity study submitted to EPA in 1986 indicates that the cancer potency level would be of about the same magnitude as the cancer potency calculated by Crump (EPA 1986c).

Mutagenicity. Numerous studies have been performed to examine the mutagenic potential of 2,4-D. Many of these studies have been reviewed by Newton and Dost (1984) and have shown negative, weakly positive, and positive results, depending upon the test systems used and the purity of the test substances. For example, microbial assays with *B. subtilis, E. coli, S. typhimurium, S. cereuisiae,* and *Bacteriophage*T4; insect chromosomal studies with *Drosophila*; cell culture studies; and mammalian chromosomal studies have all shown predominantly negative results. However, positive mutagenic responses also have been recorded in some of these test systems, including unscheduled DNA synthesis in human fibroblasts, arabain resistance in hamster lung cells, the recessive lethal assay in *Drosophila*, chromosomal aberration in mouse bone marrow, and sister chromatid exchange in human lymphocytes (Newton and Dost 1984). Furthermore, 2,4-D caused a highly significant increase in rates of sister chromatid exchange in cultured human lymphocytes at a 50 μg/mL dosage, but not at dosages of 100 and 250 μg/mL (Turkula and Jalal 1985). Bovine fetal muscle cells exposed to a culture media containing 2 and 20 mg/L of 2,4-D exhibited an initial drop in mitotic index, an increase in differentiating and degenerating cells, unipolar and tripolar spindles, and a variety of other abnormalities (Basrur et al. 1976). Also, 2,4-D induced clastogenicity (chromosomal breaks) in white rats (Turkula and Jalal 1987). According to WHO (1984), however, "studies available at present are not adequate for the quantitative evaluation of the mutagenic effects of 2,4-D and evidence does not suggest that 2,4-D derivatives are potent mutagens." Newton and Dost (1984), in their

review, concluded that 2,4-D may be a weak mutagen "but is without significance as an environmental mutagenic hazard." Thus, the review of the evidence of 2,4-D mutagenicity in this risk assessment indicates that 2,4-D cannot be ruled out as a weak mutagen, but that it is not likely to present a risk of human heritable mutations at the exposure levels that might occur in BLM's vegetation treatment program.

2,4-D Contaminants

In the case of 2,4-D, special attention must be paid to two contaminants, one of which is also a metabolic product in microorganisms. The first of these contaminants, 2,4-dichlorophenol (2,4-DCP), is an intermediate in the manufacture of 2,4-D and a minute fraction of 2,4-DCP may remain in the final product. It is also an environmental metabolite of 2,4-D. Because of its relatively low toxicity (the LD_{50} is approximately 1,300 mg/kg), however, 2,4-DCP has not been judged sufficiently toxic to be eliminated from 2,4-D formulations (Dost 1983).

The effects of 2,4-DCP on human health have not been well studied. Boutwell and Bosch (1958, as cited in Dost 1983) examined the carcinogenicity of 2,4-DCP and found it to be a very weak skin tumor promoter. It was also found to inhibit oxidative phosphorylation in rat liver and brain mitochondria (Mitsuda et al. 1963). Somani and Khalique (1982) found that after intravenous administration of 2,4-DCP in rats, the chemical was rapidly metabolized to glucuronide and other conjugates and was eliminated from the body. They showed that half-lives in the kidney and liver are longer than in other tissues, indicating that the liver is a principal organ for metabolism and that the higher levels in the kidneys correlate with that being the route of elimination. Seyler et al. (1984) performed some preliminary reproductive screening procedures and found that 2,4-DCP did not depress sperm penetration of ova and sperm motility in vitro when compared with controls. A 2,4-DCP teratology study recently reviewed by EPA found a NOEL of 350 mg/kg/day; the lowest effect level was found to be 750 mg/kg/day, with the effect being delayed ossification (EPA 1985). Nonetheless, the low toxicity of 2,4-DCP and the extremely low potential exposures of this contaminant from the parent herbicide (a worse case exposure of 0.003 mg/kg, Dost 1983) indicate that 2,4-DCP presents virtually no human health hazard.

The other impurity of concern in 2,4-D formulations is 2,7-dichloro-dibenzo-p-dioxin

(DCDD), which differs only slightly in structure from the well-known 2,3,7,8-TCDD, but differs by about a millionfold in toxicity. Two concerns of biological danger have been expressed: DCDD is alleged to be a carcinogen and a teratogen (Dost 1983).

The toxicologic studies from which these concerns arise were reported by Khera and Ruddick (1973, as cited in Dost 1983), who discussed fetotoxic effects of DCDD, and the National Cancer Institute (1979, as cited in Dost 1983), which conducted carcinogenesis studies in two species. Khera and Ruddick fed mice DCDD at dosages of 1 and 2 mg/kg daily to determine whether DCDD could cause birth defects. The observed effect at 1 mg/kg was a modest degeneration of heart muscle fibers and some fluid accumulation around the heart in a few of the animals. A somewhat greater number of animals were affected at 2 mg/kg. Both effects are in the category of general fetal toxicity. No teratogenic effect was found.

The National Cancer Institute (1979, as cited in Dost 1983) work was carried out by feeding DCDD as 0.5 and 1 percent of the total diet for 2 years. The data indicated a "suggested" carcinogenic effect in male mice that was not strong enough to support a conclusion that DCDD is a carcinogen. Neither male mice nor rats of either sex responded significantly.

DCDD has been found in 3 of 30 samples of U.S.-produced 2,4-D, along with traces of other relatively nontoxic chlorodioxins with three and four chlorines. The concentrations in the three positive samples ranged from 25 to 60 ppb. If the maximum expected human dose of 2,4-D is 0.1 mg/kg, and for convenience, all 2,4-D is assumed to contain 100 ppb of DCDD, the dose of DCDD to the exposed human would be 0.00000001 mg/kg (Dost 1983).

The conclusion, therefore, is that neither 2,4-DCP nor 2,7-DCDD, at maximum occupational or environmental exposures to 2,4-D, represents a human hazard (Dost 1983).

Data Gaps

According to EPA (1989), existing data gaps include a chronic feeding study in dogs and a teratology study in rabbits. In addition, EPA (1988) considers mutagenicity, eye irritation, dermal irritation, and 21-day dermal studies to be data gaps for 2,4-D. Lastly, partial data gaps exist for oncogenicity studies in rats and mice (EPA 1988).

Dalapon

Toxic Effects Seen in Humans

The human reference dose for chronic oral exposure to dalapon was established at 0.03 mg/kg/day by EPA (1989), based on a NOEL of 8.45 mg/kg/day in a 2-year oral toxicity study with rats. An uncertainty factor of 300 was applied to account for inter- and intraspecies variation, and to account for a significant number of data gaps for dalapon.

Threshold Toxicity in Laboratory Animals

Acute and Subacute Toxicity. Based on the lowest acute oral LD_{50} of 7,577 mg/kg in rats, dalapon can be classified as very slightly toxic. In a primary eye irritation study with rabbits, a 26.8-percent formulation of dalapon caused slight eye irritation, which subsided within 24 hours. Based on this study, EPA categorized dalapon as slightly toxic for eye irritation. The acute dermal LD_{50} for the 26.8-percent formulation in rabbits was greater than 4 g/kg (HDT). Based on the slight local erythema observed in this study, dalapon was also classified as slightly toxic for dermal toxicity (EPA 1984).

Subchronic and Chronic Toxicity. In a 2-year feeding study, rats were given 100, 300, and 1,000 ppm dalapon sodium salt in the diet. The only adverse effect noted was increased average kidney weight at the highest dose level. Microscopic examination of tissues revealed no abnormal pathology (Paynter et al. 1960, as cited in USDA 1984). The NOEL for this study was therefore established at 300 ppm (15 mg/kg/day) (EPA 1984). Because the test substance was only 65 percent dalapon, EPA has converted the NOEL to 8 mg/kg/day for 100 percent dalapon in the drinking water health advisory (EPA 1987a).

In another chronic toxicity study, dogs were administered dalapon sodium salt by capsule (15, 50, or 100 mg/kg/day) 5 days per week for 52 weeks. At the high-dose level, adverse effects were limited to an increase in average kidney weights. Histopathological examination revealed no significant difference in tissues of treated and untreated animals (Paynter et al. 1960, as cited in USDA 1984). The NOEL for this study was therefore determined to be 100 mg/kg/day (EPA 1984).

In a 2-year mouse feeding/oncogenicity study, test animals exhibited increased liver weight at 200 mg/kg/day, the highest dose tested (CDFA 1986). A systemic NOEL of 60 mg/kg/day was therefore established.

Reproductive and Developmental Toxicity. No teratogenic effects were noted in two rat teratology studies. In one study, rats were administered 500, 1,000, or 1,500 mg/kg/day by gavage during days 6 to 15 of gestation. Fetal weight was significantly decreased at 1,000 and 1,500 mg/kg (CDFA 1986). The fetotoxic NOEL was therefore established at 500 mg/kg/day, and the teratogenic NOEL was greater than 1,500 mg/kg/day (EPA 1984).

In another teratology study, pregnant rats were given 250, 500, 1,000, 1,500, or 2,000 mg/kg/day during the 6th through 15th day of gestation. Pup weights were significantly lower at the 1,000 mg/kg/day dosage level, and weight gains of pregnant dams were reduced at 1,500 mg/kg/day. No adverse effects were observed at the 250- and 500-mg/kg/day dose levels (USDA 1984).

In a three-generation reproduction study, rats were fed 0.03, 0.1, or 0.3 percent dalapon sodium salt in the diet. No adverse effects on fertility, gestation, viability, or growth and maturation were observed (Paynter et al. 1960, as cited in USDA 1984). The NOEL was reported as 0.3 percent (approximately 300 mg/kg/day) (EPA 1984). In another three-generation rat reproduction study, the NOEL was determined to be 3,000 ppm (150 mg/kg/day) (EPA 1984). A one-generation reproduction study in dogs established a NOEL of 500 ppm (12.5 mg/kg/day) (EPA 1984).

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity Study Results. In a 2-year feeding/oncogenicity study, rats were fed diets containing 100, 300, or 1,000 ppm dalapon sodium salt. At 104 weeks, histological examination of tissues revealed no differences between treated and control animals. However, findings specifically related to tumor formation were not reported (Paynter et al. 1960, as cited in USDA 1984).

In a mouse oncogenicity study reported by the California Department of Food and Agriculture (CDFA 1986), animals were fed 0, 2, 60, or 200 mg/kg/day over 2 years. Although this study was judged incomplete, CDFA (1986) concluded that dalapon was not oncogenic in this study.

No abnormal pathology or evidence of tumor formation was found of histology sections of test animal tissues in a 52-week dog feeding study (Paynter et al. 1960, as cited in USDA 1984). Thus, available data do not indicate that dalapon is carcinogenic. EPA (1987a) has placed dalapon in Group D: not classifiable as

to human carcinogenicity because of insufficient study data.

Mutagenicity. Mutagenicity studies also were reported by the California Department of Food and Agriculture (CDFA 1986). Dalapon tested negative for gene mutation in *Salmonella* with and without activation and in *Aspergillus nidulans*. Dalapon was also negative for chromosomal aberrations in the Chinese hamster ovary cell. The weight of evidence reviewed in this risk assessment therefore indicates that dalapon does not present a risk of heritable mutations.

Data Gaps

EPA (1987b) reports that data gaps for dalapon include a chronic feeding study in rats, a chronic feeding study in dogs, a reproduction study in rats, and teratology studies in rats and rabbits. In addition, no oncogenicity studies have been validated by EPA for dalapon. EPA requires oncogenicity studies in two rodent species.

The systemic NOEL for dalapon used in this risk assessment (8 mg/kg/day) was established in a chronic rat feeding study, as cited by EPA (1984); however, as stated above, an additional chronic feeding study in rats must be completed to fulfill EPA requirements.

Dicamba

Toxic Effects Seen In Humans

No human toxicity data on dicamba were available in the literature. EPA has established for dicamba a reference dose (RfD) of 0.03 mg/kg/day for chronic oral exposure based on a rabbit teratology study with a NOEL (no-observed-effect level) of 3.0 mg/kg/day (EPA 1989). An uncertainty factor of 100 was applied to the NOEL to derive the RfD.

The Pesticide Incident Monitoring System data base revealed 10 incident reports involving humans from 1966 to March 1981 for dicamba (EPA 1981, as cited in EPA 1987). Six of the ten reported incidents involved spraying operations. No concentrations were specified. Exposed workers developed symptoms that included muscle cramps, dyspnea, nausea, vomiting, skin rashes, loss of voice, and swelling of cervical glands. Coughing and dizziness resulted in one child being involved in an undescribed agricultural incident. Three children who sucked mint leaves from a ditch bank previously sprayed with dicamba were asymptomatic.

Threshold Toxicity in Laboratory Animals

Acute and Subacute Toxicity. Based on its acute oral LD_{50} of 757 mg/kg in rats, dicamba can be classified as slightly toxic (USDA 1984). Also, dicamba is very slightly toxic as a skin irritant (EPA 1984). However, dicamba is classified as a severe eye irritant (EPA 1983).

Subchronic and Chronic Toxicity. The NOEL from a 90-day rat study is given as 500 ppm (25 mg/kg/day), based on slight liver cell alterations at the 800-ppm dose (EPA 1984). In a 15-week rat feeding study, male Wistar rats (20 per dose) that were fed diets containing technical dicamba at 0, 31.6, 100, 316, 1,000, or 3,162 ppm (0, 1.6, 5, 15.8, 50, or 158 mg/kg/day) showed liver-to-body weight ratio increases at the two highest doses (EPA 1987). The NOEL for this study was determined to be 15.8 mg/kg/day.

A 2-year oral toxicity study on rats established a NOEL of greater than 125 mg/kg/day, which was the highest dose tested (EPA 1988). In addition, a 1-year oral toxicity study in dogs established a NOEL of greater than 52 mg/kg/day, which was the highest dose tested (EPA 1988).

A 90-day subchronic feeding study with male and female rats was performed with dosages of 0, 1,000, 5,000, and 10,000 ppm. The rats exhibited no compound-related changes in general behavior and appearance. The high-dose groups showed a slight decrease in comparative body weight gains and food consumption. There were no gross lesions or organ weight gain variations in treated groups. There was an absence or reduction of cytoplasmic vacuolation of hepatocyte, indicating reduced glycogen storage in high-dose groups. The no-observed-effect level was 250 mg/kg/day (systemic) (EPA 1987).

Reproductive and Developmental Toxicity. A fetotoxic NOEL of 0.5 mg/kg was reported for a rabbit teratology pilot study, with intrauterine resorptions reported at 1.0 mg/kg/day (EPA 1988). The NOEL of 0.5 mg/kg/day was not used in this risk analysis because of the supplemental EPA rating of the study and the short duration. A second rabbit teratology study resulted in a maternal NOEL and a fetotoxic NOEL of 3.0 mg/kg/day, with decreased fetal body weights and increased post implantation losses at the lowest effect level of 10 mg/kg/day. No teratogenicity was observed in this study. The NOEL of 3.0 mg/kg/day was used for the risk analysis of

dicamba. A rat teratology study revealed a maternal NOEL of 160 mg/kg/day (EPA 1988). Also, in a three-generation reproduction study, no reproductive effects were observed at 25 mg/kg/day (highest dose tested (HDT)) (EPA 1988).

Nonthreshold Toxicity in Laboratory Animals

Carcinogenicity Studies. Dicamba is not considered to be carcinogenic in this risk assessment. Although the above chronic feeding studies do not meet the current FIFRA registration guidelines, they do provide information on the chronic effects of dicamba. Likewise, although none of these studies were conducted as a cancer study (and they would not meet today's strict guidelines for cancer studies), the histopathology screening conducted does provide some information on dicamba's ability to cause cancer.

A recent 2-year rat study, accepted by EPA, showed no oncogenic or systemic effects at the highest dose tested (125 mg/kg/day) (EPA 1988). However, since dicamba's oncogenic potential has not been determined in mice, dicamba is presently included in Group D: not classifiable as to human carcinogenicity risk (EPA 1987).

Mutagenicity. Dicamba has been tested for mutagenicity and for its effect on unscheduled DNA synthesis. The following studies are cited in USDA (1984). The results were negative for gene mutation in *Salmonella typhimurium* (Poole et al. 1977, Eisenbeis et al. 1981, and Anderson et al. 1972), *Escherichia coli* (Poole et al. 1977, Andersen et al. 1972), and *Saccharomyces cerevisiae* (Poole et al. 1977) and *Bacillus subtilis* (DOE 1983). Unscheduled DNA synthesis, assayed in human fibroblast line W1-38, was negative for dicamba (Poole et al. 1977). Dicamba was positive in relative toxicity assays in *E. coli* and *B. subtilis* (Poole et al. 1977). However, the weight of evidence indicates that dicamba is not mutagenic and thus does not present a risk of heritable mutations.

Dicamba Contaminants

The manufacturing process for dicamba has the potential of resulting in traces of 2,7-dichlorodibenzo-p-dioxin as a contaminant, which is present at levels to 50 parts per billion (ppb). A more toxic dioxin isomer, 2,3,7,8-tetrachlorodibenzo-p-dioxin, has not been found at the limit of detection (2 ppb) and is not expected as an impurity in dicamba. Dicamba products formulated with diethylamine have the potential of adding

dimethylnitrosoamine (DMNA) contaminant. Nitrosoamine levels in the diethylamine formulations are expected to be less than 1 ppm. The risk levels for the dicamba products with the nitrosoamine contaminant are in the range of 1×10^{-7} to 1×10^{-5}. EPA considers the benefits to outweigh the risks associated with the nitrosoamines (EPA 1983).

Data Gaps

EPA (1989) has indicated that a data gap exists for a mouse oncogenicity study. However, dicamba is not considered oncogenic for this risk assessment because of negative results in a recent 2-year rat study that EPA (1988) reported.

Diuron

Toxic Effects Seen in Humans

EPA (1989) has established a reference dose (RfD) of 0.002 mg/kg/day, based, on the NOEL of 0.625 mg/kg/day in the dog study. An uncertainty factor of 300 was applied to account for inter- and intraspecies and to account for the lack of reproductive and developmental study data.

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. Based on the acute oral LD_{50} of 3,750 mg/kg in rats, diuron can be classified as slightly toxic (EPA 1984). Signs of toxicity were related to nervous depression and included slowed respiration and heart rate, weakness, and lethargy (EPA 1983). The LD_{50} for dermal exposure was found to be more than 10,000 mg/kg in rats; therefore, diuron is classified as very slightly toxic for dermal effects (1983). Acute primary dermal irritation studies in rabbits found slight erythema or edema at 24 hours (EPA 1983). All test data from acute primary dermal irritation studies that applied diuron to animal skin, abraded and inebriated, resulted in normal findings at 72 hours. Diuron is classified as very slightly toxic for eye irritation because no primary eye irritation was found in the unwashed eyes of rabbits (EPA 1983).

Subchronic and Chronic Toxicity. EPA (1983) evaluated two 2-year feeding studies, one using rats and one using dogs. In both of these studies, the test sample was a wettable powder formulation containing 80 percent diuron. The dietary levels were based on diuron.

In the 2-year rat study, rats were given diets containing 0, 25, 125, 250, or 2,500 ppm diuron. The investigators attributed the high mortality to an epidemic of pneumonitis-peritonitis. The highest dose depressed growth. Increased mortality was observed at the 2,500- and 250-ppm level in males given diuron. During pathology examinations, the authors noted slight anemia, enlarged spleens, increased erythrogenic activity in bone marrow, and abnormal blood pigments in the blood of groups fed 125 ppm or more. The NOEL was 25 ppm (1.25 mg/kg/day) in rats. No evidence of tumorigenicity was found. However, although this study is accepted as a chronic toxicity study, it is of only supplemental value as an oncogenicity test because limited pathology did not include all rats that died during the study or all rats sacrificed at the end of the study (EPA 1983).

A 2-year feeding study in dogs was conducted at levels of 0, 25, 125, 250, and 1,250 ppm in the diet. The highest dose caused weight loss, depressed red blood cell counts, erythrogenic activity in bone marrow, elevated liver weight, and increased pigment disposition in liver cells. Also, abnormal pigments were found in the blood of males at levels higher than 25 ppm and females at levels higher than 125 ppm. Slightly decreased hematological values were seen in the 125-ppm group, but they were statistically significant only in the red blood cell count in male dogs. No other abnormal effects were noted with respect to hematology, urine biochemistry, or histology. No evidence of tumorigenicity was found (EPA 1983). Therefore, the NOEL in dogs is 25 ppm (0.625 mg/kg/day) (EPA 1983).

Reproductive and Developmental Toxicity. In a teratology study, rats were administered 80 percent diuron by gavage from the 6th through the 15th day of pregnancy. The dose levels were 0, 125, 250, and 500 mg/kg/day. Some abnormalities were observed at all treatment levels. Among these were wavy ribs, sternoschisis, and delayed calvarium ossification, all of which could result from fetal toxicity. Delayed calvarium ossification found in one rat at the lowest dose level, 125 mg/kg/day, was of borderline significance (EPA 1983). No teratogenic effects were observed, and the teratogenic NOEL was reported as greater than 500 mg/kg (EPA 1986).

A three-generation rat reproduction study involving technical diuron resulted in body weight depression in the F_4b and F_4a litters, but this was not considered a fetotoxic, reproductive, or teratogenic effect (EPA 1984). A reproductive NOEL of greater than 125 ppm

active ingredient (6.25 mg/kg/day) (only dose tested) was established (EPA 1984).

Nonthreshold Effects In Laboratory Animals

Carcinogenicity Studies. Diuron is not considered to be carcinogenic in this risk assessment because studies relating to diuron's oncogenicity, reviewed in EPA (1983 and 1987), show no clear evidence that diuron causes tumor growth.

In a mouse oncogenicity study, 7-day-old mice were given doses of 464 mg/kg diuron by intubation for 4 weeks. After weaning, they were given diets containing 1,400 ppm diuron for 18 months. The study showed no positive evidence that diuron was tumorigenic. However, because this study was a screening study, EPA considered it to be of limited value for making final decisions (EPA 1983).

EPA (1983) also indicated that the 2-year rat and dog feeding studies, previously described as chronic studies, have only supplemental value as oncogenicity studies. Although both studies found no evidence of tumorigenicity, the limited pathology information provided in these studies does not meet EPA requirements for oncogenicity. Therefore, EPA stated, while there is no valid evidence that diuron is oncogenic, there is insufficient evidence that it is not. EPA (1983) has requested further testing of rats and another species for oncogenicity. Diuron is presently placed in Group D, not classifiable as to human carcinogenicity (EPA 1987). Therefore, diuron was not considered carcinogenic in the risk assessment.

Mutagenicity. Diuron showed negative results in microbial assays for gene mutation, DNA damage in a Chinese hamster ovary cell forward mutation assay, and in an unscheduled DNA synthesis assay in rat hepatocytes (EPA 1987). However, in an in vivo cytogenetic assay with rats, diuron caused clastogenic effects (EPA 1987). Ames assays using five strains of bacteria resulted in negative findings for mutagenicity (EPA 1987). However, in an in vivo cytogenetic assay with rats, diuron caused clastogenic effects (EPA 1987). Also, positive results were reported in a testicular DNA synthesis inhibition assay (EPA 1983). EPA (1983) believed the results of the latter test to be cause for concern because it suggests that diuron can enter the testes, and, if shown to be mutagenic, diuron may produce heritable mutagenic effects. EPA (1983) has therefore requested additional studies for chromosomal aberrations and other genotoxic effects, such as DNA damage and repair.

Because of the uncertainty in the studies and EPA's conclusion about possible entry to the testes, this risk assessment concludes that diuron may present some risk of human heritable mutations.

Data Gaps

The diuron science chapters (EPA 1983) indicate that acute inhalation (LC_{50}), dermal sensitization, oncogenicity, and mutagenicity studies are data gaps for diuron. EPA (1989) indicates that an additional reproduction study in rats must be performed because of an undetermined NOEL in the existing reproduction study. Also, additional rat and rabbit teratology studies must be submitted for diuron (EPA 1989).

The risk assessment assumes that diuron may be mutagenic because of the mutagenicity data gaps. The lowest NOEL for reproductive/developmental effects used in the risk assessment was 6.25 mg/kg/day from a three-generation rat reproduction study; however, because this dosage level was the only dose tested, EPA (1989) has requested that an additional study be performed.

Glyphosate

Toxic Effects Seen In Humans

No data for human toxicity from glyphosate are available in the current literature. A reference dose for chronic oral exposure was established at 0.1 mg/kg/day, based on a reproductive NOEL of 10 mg/kg/day with an applied uncertainty factor of 100 for intra- and interspecies variations (EPA 1989).

Threshold Effects In Laboratory Animals

Acute and Subacute Toxicity. Glyphosate can be classified as a slightly toxic chemical, based on an oral LD_{50} of 4,320 mg/kg in rats and an acute dermal LD_{50} of greater than 5,010 mg/kg in rabbits (EPA 1986a). In primary irritation studies with rabbits, glyphosate was a slight eye irritant and a very slight skin irritant (EPA 1986a).

Subchronic and Chronic Toxicity. A 26-month oral toxicity study with rats established a NOEL of 31 mg/kg/day, the highest dose tested. EPA (1988) gave this study a "minimum" rating.

A 90-day oral toxicity study in mice established a NOEL of 1,500 mg/kg/day, with reduced body weight gain at the lowest effect level of

7,500 mg/kg/day. EPA (1988) gave this study a "supplementary" rating.

A 1-year oral toxicity study in dogs revealed no effects at the highest dose tested of 500 mg/kg/day. EPA (1988) gave this study a "guideline" rating.

Reproductive and Developmental Toxicity. A three-generation reproduction study with rats established a NOEL of 10 mg/kg/day (1988). At the lowest effect level of 30 mg/kg/day, increased incidence of renal tubular dilation was observed in pups. Teratology studies established NOELs of greater than 3,500 mg/kg/day in rats and 350 mg/kg/day in rabbits (1988). EPA (1988) gave the reproduction study and the two teratology studies a "minimum" rating.

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. A 26-month rat feeding study found no oncogenic effects at doses up to 31 mg/kg/day (EPA 1986a). However, the EPA rating of this study was downgraded to "supplementary" because the maximum-tolerated dose was not reached (EPA 1986b). Benign kidney tumors (renal tubular adenomas) were found at the highest dose level (30,000 ppm) in a 2-year mouse feeding study; however, the findings were equivocal (EPA 1986b). The EPA Science Advisory Panel reviewed all relevant data, concluded that the oncogenic potential of glyphosate could not be determined from existing data, and proposed that the study be repeated to clarify these equivocal findings (EPA 1986b). Because of the uncertainty about the carcinogenicity of glyphosate, a cancer risk analysis was conducted in this risk assessment.

A carcinogenic nitrogen derivative of glyphosate, *N*-nitrosoglyphosate (NNG), is not considered a potential human hazard here because NNG is not likely to form in soils at the application rates used in forestry. Details on NNG are presented in the *Supplement to the Environmental Impact Statements on Management of Competing Vegetation* (USDI 1986).

Cancer Potency. Glyphosate's cancer potency was based on the rate of kidney tumor formation in male mice in the 2-year feeding study described above (EPA 1985). The upper 95-percent limit of the cancer potency of glyphosate calculated from the kidney tumor data was 2.4×10^{-3} per mg/kg/day.

Mutagenicity. Glyphosate was nonmutagenic in microbial assays for gene mutation and primary DNA damage and was nonmutagenic in mammalian cell assay systems, both in vitro and in vivo (EPA 1986a). There is no evidence to indicate that it is mutagenic.

Data Gaps

According to EPA (1989), oncogenicity studies are required with rats and mice for glyphosate. Although such studies were submitted previously, EPA has requested additional studies because of equivocal evidence of oncogenicity in the mouse study and the rat study's supplementary rating.

Hexazinone

Toxic Effects Seen in Humans

EPA established a reference dose of 0.033 mg/kg/day for chronic oral exposure based on a 2-year oral rat toxicity with a NOEL of 10 mg/kg/day (1989). An uncertainty factor of 300 was used to derive the RfD.

The Pesticide Incident Monitoring System data base (EPA 1981, as cited in EPA 1987) indicated that 3 of 43,729 incident reports involved hexazinone. Only one report cited exposure to hexazinone alone, with no other compounds involved. A 26-year-old woman inhaled hexazinone dust. Vomiting occurred within 24 hours. No other effects were reported and no treatment was administered. No concentration was specified for this incident.

Acute and Subacute Toxicity. Hexazinone is classified as slightly toxic based on the acute oral LD_{50} of 1,690 mg/kg and the acute dermal LD_{50} of 5,278 mg/kg (EPA 1986). Acute toxicity effects include pallor, salivation, nose bleeds, dyspnea, lethargy, tremors, and convulsions (USDA 1984). Although hexazinone is a very slight skin irritant, it is classified as a severe eye irritant (EPA 1988).

Subchronic and Chronic Toxicity. In a 90-day rat feeding study, the only observed effect was reduced body weight gain at a dosage of 250 mg/kg/day (HDT) (EPA 1988). Slight liver effects and reduced body weight gain were noted in dogs at 125 mg/kg/day in a 3-month feeding study (EPA 1988).

A NOEL of 30 mg/kg/day was established in a 2-year oral toxicity study in mice. Toxic effects observed during the mouse study included increased liver size, a localized increase in size and number of liver cells, and

localized tissue degeneration at the lowest effect level (LEL) of 375 mg/kg/day (EPA 1986). A 2-year oral toxicity study in rats established a NOEL of 10 mg/kg/day. Effects observed in rats included reduced body weight gain, decreased food consumption, increased leukocyte counts, and excretion of a more alkaline urine (EPA 1988).

Reproductive and Developmental Toxicity. In a three-generation reproduction study, no effects on reproduction performance or lactation were observed in rats at the highest dose (125 mg/kg/day); however, the average body weight of pups at weaning was slightly lower at this dosage level (EPA 1988). Thus, the reproductive NOEL was established at 125 mg/kg/day, and the fetotoxic NOEL was established at 50 mg/kg/day. In rat teratology study, pregnant rats were fed hexazinone by garbage once daily on days 7 to 16 of gestations. Doses of 0, 40, 100, 400, and 900 mg/kg/day were administered. Based on decreased female fetal weight, marginally increased kidney anomalies, and an increase in unossified sternebrae in fetuses, a teratogenic NOEL of 100 mg/kg/day was established (EPA 1988). However, in another rat teratology study, no developmental effects were observed up to 250 mg/kg/day, the highest dose tested (EPA 1988). Similarly, no teratogenic effects were observed in rabbits at 125 mg/kg/day (HDT) in a teratology study (EPA 1988).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. Available evidence indicates that hexazinone is not carcinogenic. In 2-year mouse and rat feeding studies, no oncogenic effects of hexazinone were observed at any of the doses tested (10, 50, and 125 mg/kg/day in rats and at the testing levels of 30, 375, and 1,500 mg/kg/day in mice) (USDA 1984, EPA 1986). Hexazinone is considered noncarcinogenic for this risk assessment.

Mutagenicity. Hexazinone was nonmutagenic in Ames assays, in an in vitro mammalian point mutation assay, in an assay of unscheduled DNA repair synthesis in mammalian somatic cells, and in an in vivo mammalian cytogenetic assay (EPA 1986, USDA 1984). Hexazinone induced chromosome damage in an in vitro cytogenetic assay with Chinese hamster ovary cells both with and without metabolic activation (EPA 1986). However, this effect was observed only at very high levels. Based on these results, hexazinone is considered nonmutagenic to humans for this risk assessment.

Data Gaps

According to EPA (1989), a chronic dog study is the only existing data gap for hexazinone. However, EPA (1988) also lists acute oral, acute dermal, and acute inhalation studies as partial data gaps and a 21-day dermal study as a data gap.

Imazapyr

Health Effects Seen in Humans

No EPA reference dose has been established for imazapyr.

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. Based on an acute oral LD_{50} of greater than 5,000 mg/kg in rats, imazapyr is considered very slightly toxic to mammals (EPA 1985). The acute dermal LD_{50} of imazapyr is greater than 2,000 mg/kg body weight in both rats and rabbits (EPA 1985, American Cyanamid 1985). In primary irritation studies, imazapyr was slightly irritating to the eyes and very slightly irritating to rabbits' skin. A dermal sensitization test was negative in guinea pigs. A 21-day dermal study in rabbits showed no signs of systemic toxicity at 400 mg/kg/day (EPA 1985).

Subchronic and Chronic Toxicity. American Cyanamid (1985) reported a 13-week rat feeding study that established a NOEL of 500 mg/kg/day (HDT).

Reproductive and Developmental Toxicity. A maternal toxic NOEL of 300 mg/kg/day was established in a rat teratology study based on salivation at 1,000 mg/kg/day (EPA 1985, American Cyanamid 1985); however, no teratogenic or fetotoxic effects were observed in rats at 1,000 mg/kg (the highest dose tested). In addition, no effects were observed at a dosage level of 400 mg/kg/day in a rabbit teratology study (EPA 1985, American Cyanamid 1985).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. No evidence of carcinogenicity was observed within the first 12 months of a chronic feeding/oncogenicity study in rats at the highest dosage level of 500 mg/kg/day, the highest dose tested (Biodynamics, Inc., undated). Further study results must be obtained before the carcinogenic potential of imazapyr can be determined.

Mutagenicity. Imazapyr was nonmutagenic in the Ames bacterial assays (with and without metabolic activation), the dominant lethal mouse assay, a Chinese hamster ovary in vitro cytogenetic assay, an unscheduled DNA repair synthesis test, and the Chinese hamster ovary cell HGPRT assay (gene mutation mammalian germ cell test) (American Cyanamid 1985, 1986). Based on these results, imazapyr is determined to be nonmutagenic for this risk assessment.

Data Gaps

Although no evaluation of the EPA data base for imazapyr is currently available, data gaps for imazapyr appear to include a reproduction study in rats and chronic oral toxicity studies in rats and dogs.

The NOEL established in a 13-week oral toxicity study was used as the systemic NOEL in this risk assessment. The reproductive NOEL used in this risk assessment was established in a rabbit teratology study.

Mefluidide

Toxic Effects Seen In Humans

No human toxicity data on mefluidide were available in the literature.

Threshold Effects In Laboratory Animals

Acute and Subacute Toxicity. Based on an acute oral LD_{50} of greater than 4,000 mg/kg for rats and 1,920 mg/kg for mice (WSSA 1983), mefluidide is classified as slightly toxic. The acute inhalation LC_{50} for rats was found to be greater than 8.5 mg/L/4 hours (WSSA 1983). The dermal LD_{50} for rabbits was found to be greater than 4,000 mg/kg (WSSA 1983). A primary dermal irritation study in rabbits showed that mefluidide produced no irritation to either abraded or unabraded skin (WSSA 1983). A primary eye irritation study revealed that mefluidide caused minimal irritation in rabbits (3M 1987).

Subchronic and Chronic Toxicity. Subchronic oral toxicity tests with mefluidide were carried out in rats and dogs. Ninety-day dietary feeding studies in these animals established a systemic NOEL of 6,000 ppm (300 mg/kg/day) for rats and 1,000 ppm (25 mg/kg/day) for dogs (WSSA 1983).

Chronic toxicity tests with mefluidide were carried out in mice, rats, and dogs. In mouse 18-month and rat 2-year dietary feeding studies, groups of mice and rats were fed 0,

600, 1,800, or 6,000 ppm mefluidide in their diets (EPA 1988). These studies yielded a systemic NOEL of 600 ppm (90 mg/kg/day) for mice and 600 ppm (30 mg/kg/day) for rats.

In a 1-year feeding study, dogs were fed mefluidide at the dietary levels of 0, 60, 600, or 6,000 ppm (EPA 1988). Dogs in the 600-ppm group showed cortical nephrosis. Based on this adverse effect, EPA (1988) established a NOEL of 60 ppm (1.5 mg/kg/day) for mefluidide.

Reproductive and Developmental Toxicity. A three-generation reproduction study with mefluidide was conducted in rats (EPA 1988). The pups in the 6,000-ppm group showed reduced body weight, liver weight, and spleen weight. Based on these adverse effects, EPA established a reproductive NOEL of 1,800 ppm (90 mg/kg/day) for mefluidide.

Teratogenicity studies with mefluidide were carried out in rabbits and rats (EPA 1988). These studies established a teratogenic NOEL of greater than 60 mg/kg/day (the highest dose tested) for both rabbits and rats.

Nonthreshold Effects In Laboratory Animals

Carcinogenicity Studies. Available data do not indicate that mefluidide is carcinogenic. In the mouse 18-month and rat 2-year dietary feeding/carcinogenicity studies described above, carcinogenic effects were not found at dose levels up to 6,000 ppm (900 mg/kg/day) for mice and 6,000 ppm (300 mg/kg/day) for rats (EPA 1988).

Mutagenicity. Mefluidide was nonmutagenic in the following six assays that EPA (1988) reported in the tox one-liner on mefluidide: three Ames bacterial assays, DNA damage/repair assay, lymphoma mutation assay, and sister chromatid exchange assay.

Data Gaps

No evaluation of the EPA data base for mefluidide is currently available. Based on review of the EPA (1988) tox one-liner, there do not appear to be any existing data gaps to fulfill the registration requirements of this herbicide.

Metsulfuron Methyl

Toxic Effects Seen In Humans

EPA established a reference dose (RfD) for metsulfuron methyl of 0.25 mg/kg/day for chronic oral exposure based on a 2-year oral

toxicity study in rats with a NOEL of 25 mg/kg/day (EPA 1989). An uncertainty factor of 100 was applied to the NOEL to derive the RFD.

Threshold Effects In Laboratory Animals

Acute and Subacute Toxicity. Based on an acute oral LD_{50} of greater than 5,000 mg/kg in rats (EPA 1988b), metsulfuron methyl is classified as very slightly toxic. The acute dermal LD_{50} for rabbits was found to be greater than 2,000 mg/kg, and a primary dermal irritation study showed that metsulfuron methyl (70 percent formulation) was moderately irritating in rabbits (EPA 1988b). The acute inhalation LC_{50} for rats was found to be greater than 5.0 mg/L/4 hours (Du Pont 1984). In a primary eye irritation study in rabbits, instillation of metsulfuron methyl into rabbits' eyes induced slight corneal clouding, moderate iritis, and severe to moderate conjunctivitis in unwashed eyes (Du Pont 1984). All effects were reversed within 14 days.

Subchronic and Chronic Toxicity. Subchronic oral toxicity tests with metsulfuron methyl were carried out in rats and dogs. A 90-day feeding study in rats established a systemic NOEL of 1,000 ppm (50 mg/kg/day), based on a reduction in body weight gains and serum protein in the 7,500-ppm group (EPA 1988b). A 90-day feeding study in dogs resulted in a systemic NOEL of greater than 5,000 ppm (125 mg/kg/day), the highest dose tested (Du Pont 1984).

Chronic oral toxicity tests with metsulfuron methyl were carried out in rats, mice, and dogs. Groups of rats and mice were fed 0, 5, 25, 500, or 5,000-ppm metsulfuron methyl in the diet for 2 years and 18 months, respectively (EPA 1988b). Rats in the 5,000-ppm group showed a reduction in body weight gains. Based on this adverse effect in rats, EPA established a systemic NOEL of 500 ppm (25 mg/kg/day) for metsulfuron methyl. In the mouse study, the systemic NOEL was determined to be greater than 5,000 ppm (750 mg/kg/day), the highest dose tested. EPA (1988b) gave this 2-year feeding study in mice a "supplementary" rating because of an equivocal maximum tolerated dose in the mouse study. In a 1-year feeding study, dogs were fed metsulfuron methyl at dietary levels of 0, 50, 500 or 5,000 ppm (EPA 1988b). This study established a systemic NOEL of 50 ppm (1.25 mg/kg/day) for metsulfuron methyl, based on a reduction in serum lactate dehydrogenase activity in the 500-ppm group.

Reproductive and Developmental Toxicity. In a two-generation rat reproduction study, in which rats were fed 0, 25, 500, or 5,000 ppm metsulfuron methyl, both the reproductive and fetotoxic NOELs were determined to be greater than 5,000 ppm (250 mg/kg/day) (EPA 1988b). This study resulted in a maternal NOEL of 500 ppm (25 mg/kg/day), based on a reduction in body weight gains in the 5,000-ppm group.

Teratogenicity tests with metsulfuron methyl were carried out in rats and rabbits. A rat teratogenicity study established a fetotoxic and teratogenic NOEL of 1,000 mg/kg/day, the highest dose tested (Du Pont 1984). In a rabbit teratogenicity study, a fetotoxic and teratogenic NOEL of 700 mg/kg/day (the highest dose test) was reported, as well as a maternal NOEL of 25 mg/kg/day (EPA 1988b).

Nonthreshold Effects In Laboratory Animals

Carcinogenicity Studies. Available data do not indicate that metsulfuron methyl is carcinogenic. In the mouse 18-month and rat 2-year feeding/carcinogenicity studies, carcinogenic effects were not observed at dose levels up to 5,000 ppm (750 mg/kg/day) for mice and 5,000 ppm (250 mg/kg/day) for rats (EPA 1988b). Based on EPA's Integrated Risk Information System (EPA 1988a), EPA has not evaluated this chemical for evidence of human carcinogenic potential.

Mutagenicity. Metsulfuron methyl was nonmutagenic in an Ames bacterial assay, a Chinese hamster ovary cell assay, a DNA rat liver repair assay, and an in vivo rat bone marrow cytogenetic assay. However, the herbicide was mutagenic in an in vitro Chinese hamster ovary cell cytogenetic assay (Du Pont 1984). Therefore, metsulfuron methyl is not considered to be a potential human mutagen in this risk assessment.

Data Gaps

EPA (1988a, 1989) reported that there are no data gaps for metsulfuron methyl.

Picloram

Toxic Effects Seen In Humans

EPA established a reference dose of 0.07 mg/kg/day, based on a 6-month oral dog study with a NOEL of 7 mg/kg/day and an uncertainty factor of 100 (EPA 1989).

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. With an acute oral LD_{50} of 4,012 mg/kg in female rats (EPA 1988a), picloram is classified as slightly toxic. Slight eye and very slight skin irritation was observed in primary eye and primary dermal irritation studies using rabbits (EPA 1988a). The acute dermal LD_{50} is greater than 2,000 mg/kg (1988a).

Subchronic and Chronic Toxicity. A 6-month dog feeding study, during which test animals were exposed to picloram at the dietary levels of 0, 7, 35, and 175 mg/kg/day, resulted in a subchronic NOEL of 7 mg/kg/day (Barna-Lloyd et al. 1982, as cited in Mullison 1985; EPA 1988a). Increased liver weights were reported at the lowest effect level of 35 mg/kg/day in males. Another subchronic feeding study resulted in slight liver effects at 150 mg/kg/day in rats (EPA 1988a).

In a recent 2-year chronic toxicity/oncogenicity study reported by Dow (1987), rats fed 20 mg/kg/day showed no treatment-related effects. Rats given 60 and 200 mg/kg/day exhibited increased size and altered properties of liver cells. No other chronic feeding studies have been reported; EPA has requested a chronic nonrodent feeding study for picloram (EPA 1988a).

Reproductive and Developmental Toxicity. A three-generation rat reproduction study resulted in a NOEL of 50 mg/kg/day, based on reduced fertility at the highest dose tested of 150 mg/kg/day (EPA 1988b). In a rat teratology study, maternal toxicity was observed at 750 mg/kg (EPA 1988a). Some fetotoxicity (delayed bone ossification) was also noted in this study. However, no fetotoxic NOEL was determined and EPA has requested a repeat teratology in rats (1988a). No dose-related embryotoxic or teratogenic responses were observed in rabbits given doses of picloram of up to 400 mg/kg/day (John-Greene et al. 1985).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. Picloram was not oncogenic in mice at a dose of 750 mg/kg (HTD) (EPA 1988b). However, the herbicide was weakly oncogenic in female rats at a dosage of 744 mg/kg/day (HTD), based on the incidence of hepatic nodules in the liver (EPA 1988b). The oncogenic NOEL for this study was established to be 372 mg/kg/day. Although EPA (1988b) rated the study as "supplementary" and placed picloram on the Group D oncogen list (not classifiable as to

human carcinogenicity) (EPA 1988a), the herbicide is considered oncogenic in this risk assessment. The cancer potency value used in the analysis is 0.003 per (mg/kg/day) (EPA 1988b).

Mutagenicity. Picloram was nonmutagenic in an Ames assay (with and without activation), a cytogenetic bone marrow study with rats, an assay with *Aspergillus nidulans*, and an assay with *Salmonella typhimirium* (EPA 1988b). However, picloram was mutagenic in a forward mutation assay with *Streptomyces coelicolor* (EPA 1988b, CDFA 1986). Thus, based on the available evidence, picloram is considered nonmutagenic in this risk assessment.

Data Gaps

According to EPA (1988a, 1989), picloram data gaps exist for 21-day dermal rat and mouse oncogenicity, teratology, mutagenicity, and reproduction studies.

The reproductive NOEL in this risk assessment was taken from a three-generation rat reproduction study; however, the study was rated "supplementary" by EPA, and submission of an additional study is required. Picloram is considered carcinogenic in this risk assessment because of liver tumors produced in female rats (EPA 1988b). Although oncogenicity studies with rats and mice were previously submitted, EPA has requested that additional studies be performed because of the "supplementary" ratings of the previous studies (EPA 1988a, 1989).

Simazine

Toxic Effects Seen in Humans

EPA has established a reference dose (RfD) for simazine of 0.002 mg/kg/day for chronic oral exposure, based on a 2-year dietary oncogenicity study with a NOEL of 0.52 mg/kg/day (10 ppm)(1989b). An uncertainty factor of 300 was applied to the NOEL to derive the RfD.

Yelizarov (1977, as cited in EPA 1987a) noted 124 cases of contact dermatitis in the Soviet Union among workers manufacturing simazine and propazine. Mild cases lasting 3 or 4 days involved pale pink erythema and slight edema. Serious cases lasting 7 to 10 days involved greater erythema and edema and also a vesiculopapular reaction that sometimes progressed to the formation of bullae.

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. Based on the acute oral LD_{50} of greater than 5,000 mg/kg/day in rats, simazine is classified as very slightly toxic (1987b). Based on available data, EPA has classified simazine as moderately toxic for acute inhalation toxicity (EPA 1987b). A primary eye irritation study with simazine produced only a transient inflammation of the conjunctiva but no irritation to the iris or cornea in rabbits (USDA 1984). The dermal LD_{50} for simazine was established to be greater than 10,000 mg/kg (EPA 1983). A 21-day subacute dermal toxicity study in rabbits at doses of up to 1,000 mg/kg/day produced no systemic toxicity and no dose-related alterations of the skin. The findings of this study indicate a NOEL of more than 1,000 mg/kg/day (EPA 1983).

Subchronic and Chronic Toxicity. In a 3-week rat feeding study, test animals were given dose levels of 200, 2,000, or 4,000 ppm of technical simazine. At the lowest dose tested, rats exhibited reduced erythrocyte and leucocyte counts and elevated cholesterol and inorganic phosphate levels. The maximum tolerated dose (MTD) was determined to be less than 2,000 ppm (100 mg/kg/day) because this dose seriously affected the nutrition of treated rats. The NOEL was less than 200 ppm (10 mg/kg/day) (EPA 1987b).

In a 3-week dog feeding study, beagles were given simazine doses of 200, 2,000, or 4,000 ppm. At the 2,000-ppm (50 mg/kg/day) level, reduced albumin levels, increased globulin levels, and elevated urinary specific gravity and ketone levels were observed. The MTD was also reported as less than 2,000 ppm based on the seriously affected nutrition of treated dogs. The NOEL for this study was set at 200 ppm (5 mg/kg/day) (EPA 1987b).

In a 22-week feeding study with sheep, adverse effects were observed at the lowest dose tested, 1.4 mg/kg/day (EPA 1987a). However, because of deficiencies in the study, this NOEL was not used as the lowest NOEL to determine the reference dose. A 2-year chronic rat feeding study showed no dose-related pathological changes at 1-, 10-, and 100-ppm dose levels (EPA 1983, 1987b). Mortality was primarily the result of respiratory infections, with very few males in test and control groups surviving (EPA 1983). Histopathologic evaluation was not provided for animals that died during the study (EPA 1983). The systemic NOEL for simazine in this study was greater than 100 ppm (5 mg/kg/day) (EPA 1987b).

A 2-year chronic dog feeding study showed no signs of toxicity from simazine dosages of 15, 150, and 1,500 ppm, except net weight loss at 1,500 ppm and lower weight gain at 150 ppm (EPA 1983). Weight gain differences did not occur during the second half of the study. The ages of individual dogs used in the study and individual observation records were lacking. Chronic toxicity could not be determined from this study (EPA 1983).

Reproductive and Developmental Toxicity. In a rabbit teratology study, New Zealand white rabbits were administered 5, 75, or 200 mg/kg/day simazine by gavage. Doses at the 75-mg/kg/day level caused tremors, abortions, and decreased body weight gain and food consumption. At the 200-mg/kg/day level, reduced mean fetal weight and increased skeletal variations were observed. No teratogenic effects were noted at the highest dose tested (200 mg/kg/day). The NOELs reported for this study were as follows: 5 mg/kg/day for maternal effects, 75 mg/kg/day for fetotoxic effects, and greater than 200 mg/kg/day for teratogenic effects (EPA 1987b).

In a three-generation reproduction study, simazine had no adverse effects on reproductive performance in rats at a dietary level of 100 ppm. The findings of this study indicate a NOEL of greater than 100 ppm (5 mg/kg/day) (EPA 1983).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. In a two-year dietary oncogenicity study, rats were tested at 0, 10, 100, and 1000 mg/kg doses. A significant increase in mammary gland carcinomas was observed in females at 100 and 1000 mg/kg, while an increased incidence of liver tumors was noticed in moles at the same doses (EPA 1989a). Although a ninety-five week oncogenicity feeding study in mice found no significant increase in tumors up to and including the highest dose of 4000 mg/kg, EPA has concluded that the weight of evidence suggests a possible oncogenic potential for simazine (EPA 1989a). Consequently, simazine has been classified as a possible human carcinogen by EPA, and accordingly, the herbicide is considered to be carcinogenic in this risk assessment. The cancer potency value used in this risk analysis, calculated from the rat two-year study, is 0.083 per mg/kg/day.

Mutagenicity. In studies reviewed by EPA (1987a, 1989a) simazine was nonmutagenic in microbial assays with *Salmonella typhimurium*,

Escherichia coli, Bacillus subtilis, Serratia marcescens, and *Saccharomyces cerevisiae.* Simazine induced lethal mutations in a sex-linked recessive lethal test with *Drosophila melanogaster* (fruitflies) and also increased x-linked lethal with male *D. melanogaster* (EPA 1987a). In addition, simazine tested positive in an unscheduled DNA synthesis assay with human lung fibroblasts (EPA 1987a) and in a mouse lymphoma assay (EPA 1989a). However, a similar study with human lung fibroblasts (EPA 1987a) and an unscheduled DNA synthesis assay with rat hepatocytes (CDFA 1986) showed negative responses. Finally, simazine did not produce chromosomal effects in a sister-chromatid exchange test (EPA 1987a). The available evidence thus indicates that simazine may have some genotoxic potential (EPA 1989a) and is considered mutagenic in this risk assessment.

Data Gaps

EPA (1989b) considers rat reproduction and teratology studies to be data gaps for simazine. Also, data gaps exist for mutagenicity (EPA 1989).

Sulfometuron Methyl

Toxic Effects Seen in Humans

No information was available on the toxicity of sulfometuron methyl to humans.

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. Sulfometuron methyl is very slightly toxic, based on an acute oral LD_{50} of greater than 5,000 mg/kg in rats (EPA 1984). In acute dermal studies, an LD_{50} of greater than 2,000 mg/kg was reported (EPA 1984). Reversible eye and skin irritation was observed in primary eye and primary dermal irritation studies using rabbits (EPA 1984).

Subchronic and Chronic Toxicity. A 90-day rat feeding study established a systemic NOEL of 50 mg/kg/day, based on hematological effects observed at 250 mg/kg/day (EPA 1984). A 2-year rat feeding study reported by Du Pont (1986) established a systemic NOEL of 2.5 mg/kg/day. In this study, hemolytic effects, liver toxicity, and decreased brain weights were observed at 250 mg/kg/day. Hemolytic effects and liver toxicity were also observed at 25 mg/kg/day. In a 1-year dog feeding study, a systemic NOEL of 5 mg/kg/day was reported (EPA 1984). Effects observed in dogs included decreased number of red blood cells and increased liver weight at 25 mg/kg/day.

Reproductive and Developmental Toxicity. In a rat reproduction study, a NOEL of 25 mg/kg/day was established, based on decreased body weights and reduced numbers of offspring (Du Pont 1986). A one-generation rat reproduction study resulted in the establishment of a reproductive NOEL of greater than 250 mg/kg/day (HDT) (EPA 1984). A rat teratology feeding study reported reduced body weight gain at 250 mg/kg/day and maternal and fetotoxic NOELs of 50 mg/kg/day (EPA 1984). No teratogenic effects were observed at 250 mg/kg/day, the highest dose tested. A rabbit teratology study was negative for teratogenic, maternal, and fetotoxic effects at 300 mg/kg, the HDT (EPA 1984).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. No oncogenic effects were reported from a 2-year rat feeding or in 1-year chronic dog feeding studies (Du Pont 1986). Based on these data, sulfometuron methyl is not considered carcinogenic for this risk assessment.

Mutagenicity. Sulfometuron methyl was nonmutagenic when tested in an activated *Salmonella* assay (bacteria gene mutation test) and a Chinese hamster ovary cell assay (mammalian germ cell test) (EPA 1984). Du Pont (1986) also reported negative results for in vitro cytogenetic and unscheduled DNA synthesis assays in mammals. Based on these results, sulfometuron methyl is considered nonmutagenic for this risk assessment.

Data Gaps

EPA has not yet reviewed the data gaps for sulfometuron methyl. Several areas were identified in researching this hazard analysis for which information was unavailable. These data gaps include an acute rat inhalation study, a guinea pig dermal sensitization study, 21- and 90-day dermal studies, 90-day inhalation and neurotoxicity studies, and a mouse oncogenicity study. However, sufficient data were available from existing information to conduct a quantitative risk analysis.

Tebuthiuron

Toxic Effects Seen in Humans

EPA established a reference dose of 0.07 mg/kg/day for chronic oral exposure based on a two-generation reproduction study in rats with a NOEL of 5 mg/kg/day (1989). An uncertainty factor of approximately 100 was used to derive the RfD.

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. No EPA-validated studies exist for assessing acute oral toxicity of tebuthiuron (EPA 1987). The lowest rat acute oral LD_{50} that USDA (1984) reported as 644 mg/kg. Based on this study, tebuthiuron is classified as slightly toxic. Tebuthiuron induced slight eye irritation in rabbits but no skin irritation during primary eye and primary dermal studies (EPA 1986). However, tebuthiuron induced skin irritation in an acute dermal study with rabbits (EPA 1986). The LD_{50} for this study was greater than 200 mg/kg.

Subchronic and Chronic Toxicity. A systemic NOEL of 12.5 mg/kg/day was established in a 3-month dog feeding study, based on increased thyroid and spleen weight (EPA 1986). Toxic effects in other subchronic studies included growth suppression and pancreatic lesions at 125 mg/kg/day in rats and body weight depression at 37.5 mg/kg/day in cattle (EPA 1986).

A systemic NOEL of 25.0 mg/kg/day was established in a 1-year dog feeding study (EPA 1987). Effects observed at 50 mg/kg/day (the highest dose tested) included increased liver-to-body-weight ratios in both sexes, increased kidney-to-body-weight ratios in females, and increased thyroid-to-body-weight ratios in males. In addition, significantly increased liver enzyme levels were observed in males and females at 50 mg/kg/day.

In a 2-year feeding oncogenicity study in rats, a systemic NOEL was established at 20 mg/kg/day, based on growth suppression at a dosage level of 40 mg/kg/day (the highest dose tested)(EPA 1986). A 2-year feeding/oncogenicity study in mice revealed no adverse effects at the highest dose tested of 240 mg/kg/day (EPA 1986).

Reproductive and Developmental Toxicity. In a two-generation rat reproduction study, first-generation female rats at dosage levels of 10 and 20 mg/kg/day exhibited a lower rate of body weight gain as compared to the concurrent control group. No other adverse effects were observed in this study. Based on these results, a NOEL for reproductive effects was established at 20 mg/kg/day (the highest dose tested). The maternal NOEL was established at 5 mg/kg/day (EPA 1987).

A teratology study with rabbits revealed no effects at 25 mg/kg/day (EPA 1986).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. Available evidence does not indicate that tebuthiuron is carcinogenic. In 2-year rat and mouse oncogenicity studies, no oncogenic effects were observed at oral doses up to 80 and 240 mg/kg/day (the highest doses tested), respectively (EPA 1986). For the purpose of this risk assessment, tebuthiuron is considered nononcogenic.

Mutagenicity. Tebuthiuron was nonmutagenic when tested with and without metabolic activation in bacterial assay systems, in a dominant lethal rat assay, and in an activated mouse lymphoma cell assay. It was mildly mutagenic in a mouse lymphoma somatic cell test without metabolic activation (EPA 1986). Based on the battery of tests performed, tebuthiuron is assumed to be nonmutagenic.

Data Gaps

According to the tebuthiuron toxicology chapter (EPA 1987), EPA considers acute oral (rat), acute dermal, eye irritation (rabbit), dermal irritation (rabbit), dermal sensitization (guinea pig), chronic toxicity (rodent), teratology mutagenicity, and oncogenicity studies to be data gaps for tebuthiuron. The chronic rat and teratology studies, previously submitted to EPA, must be repeated because of supplemental ratings (EPA 1989).

Triclopyr

Toxic Effects Seen in Humans

The Office of Pesticide Programs established a reference dose for chronic oral exposure of 0.025 mg/kg based on a 6-month oral toxicity study in dogs with a NOEL of 2.5 (EPA 1989). An uncertainty factor of 100 was applied.

Threshold Effects in Laboratory Animals

Acute and Subacute Toxicity. With an acute oral LD_{50} ranging from 630 to 729 mg/kg in rats (EPA 1986), triclopyr is classified as slightly toxic. However, toxicities for different mammalian species may vary, as demonstrated by the moderately toxic effect of triclopyr on guinea pigs (LD_{50} = 310 mg/kg, EPA 1985c). Acute and primary dermal tests in rabbits revealed that triclopyr was slightly irritating to the rabbits' skin (EPA 1986). A primary eye irritation test demonstrated that triclopyr was moderately irritating to rabbits' eyes (EPA 1986).

Subchronic and Chronic Toxicity. A 90-day rat feeding study established a NOEL of 30 mg/kg/day based on decreased body weight, food consumption, and absolute liver weights at a dosage level of 100 mg/kg/day (EPA 1986). A 2-year feeding/oncogenic study with rats revealed no effects at dosage levels up to 30 mg/kg/day (HDT) (EPA 1986). In a 2-year feeding/oncogenicity study reported by Dow (1987), no toxicological effects were observed in rats at 3 mg/kg/day. Male rats fed 12 and 36 mg/kg/day had increased absolute and relative kidney weights.

A 228-day dog feeding study resulted in a systemic NOEL of less than 5 mg/kg/day based on phenolsulfonphthalein excretion (EPA 1986). A 6-month feeding study with dogs resulted in the establishment of a systemic NOEL of 2.5 mg/kg (HDT) (40 CFR Part 180 50(84):184-85, May 1, 1985). The effects observed in the dog studies are not representative of effects expected in humans because dogs have a limited capacity for organic anion transport in the kidney (Dow 1985). Dogs excrete triclopyr at a slower rate than other laboratory animals or humans. The half-life of triclopyr for urinary excretion in dogs is 96 hours, compared to 1.5 hours in rats and 3.1 hours in monkeys. Dow concluded that toxicity may be increased in dogs because of the greater relative retention time of the compound in the animal's body. Based on the Dow conclusions regarding this study, the use of the 2.5 mg/kg/day NOEL from the dog study (the lowest NOEL found in current available literature) in this risk assessment may be very conservative and may tend to overestimate expected effects in humans with normal renal function.

Reproductive and Developmental Toxicity. In a rat teratology study, test animals were given doses of 0, 50, 100, or 200 mg/kg/day by gavage during days 6 through 15 of gestation. No teratogenic effects were noted at the highest dose tested. Retarded ossification of skull bones was observed at 200 mg/kg/day, and decreased body weight gains and food consumption were found at 50 mg/kg/day. Therefore, the fetotoxic NOEL was established at 50 mg/kg/day, and the maternal toxic NOEL was established at less than 50 mg/kg/day (EPA 1986).

In a three-generation reproduction study, rats were given doses of 0, 3, 10, and 30 mg/kg/day in the diet. No effects were noted at the highest dose tested (EPA 1986).

Two rabbit teratology studies were conducted with triclopyr. In one study, rabbits were given 0, 10, or 25 mg/kg/day by gavage during days 6 through 18 of gestation. Reduced body weight values were observed at the 25-mg/kg/day level. The NOEL for this study was established at 10 mg/kg/day (EPA 1988). In the other study, no teratogenic effects were noticed up to the highest dose tested, 100 mg/kg/day (EPA 1986).

Nonthreshold Effects in Laboratory Animals

Carcinogenicity Studies. Available data do not indicate that triclopyr is carcinogenic. For both rat and mouse 2-year feeding studies, no oncogenic effects were apparent in test animals exposed to triclopyr (30 and 36 mg/kg/day, respectively) (EPA 1986, 40 CFR Part 180 50(84):18485-86, May 1, 1985). A recent 2-year chronic toxicity/oncogenicity study in rats has been submitted in response to EPA's request for a repeat rat oncogenicity study (Dow 1987). A statistically significant increase in mammary tumors was observed when the number of adenomas (one) and adenocarcinomas (four) were combined for high-dose females (36 mg/kg/day) (Dow 1987). However, the researchers reported that the incidence was within a range of historical controls and the statistical result was partially because of the low incidence (zero) in control rats. Based on these results, triclopyr is not considered carcinogenic for this risk assessment.

Mutagenicity. Except for a dominant lethal rat assay in which weakly positive results were observed, triclopyr was nonmutagenic in various test systems, including bacteria assays, a dominant lethal mouse assay, a cytogenetic mammalian assay in vivo, a host mediated assay in mice, and an unscheduled DNA synthesis assay with rat hepatocytes (EPA 1988). Therefore, triclopyr is not considered a potential human mutagen in this risk assessment.

Data Gaps

EPA (1989) indicates that data gaps for triclopyr have not yet been identified.

Toxicity of Herbicide Carriers

Petroleum Distillates (Diesel Oil and Kerosene)

Threshold Effects

With an acute oral LD_{50} of 9.0 mL/kg (7,380 mg/kg)(1 milliliter of diesel oil weighs 820 milligrams), diesel oil is classified as very

slightly toxic (Beck et al. 1982). The most marked acute toxic effect observed after the administration of diesel oil to test animals occurred during primary dermal irritation studies (Beck et al. 1982). In these studies, a single exposure of rabbits to diesel oil resulted in a rating of "extremely irritating," based on a score of 6.81 (on a scale of 1 to 10). The irritation may have been caused by additives for internal combustion in diesel oil. Diesel oil was nonirritating in primary eye irritation studies (Beck et al. 1982). A subacute 3-week dermal study of eight rabbits reported an average weight loss of 0.30 kilograms at the dose level of 4.0 mL/kg (3,280 mg/kg) and an average weight loss of 0.55 kilograms, with a 67-percent mortality rate at the dose level of 8.0 mL/kg (6,560 mg/kg) (Beck et al. 1982). An inhalation teratology study in which rats were exposed to 5.09 or 20.075 uL/kg of diesel fuel on days 6 through 15 of gestation did not result in any significant teratogenic effects (Mecler and Beliles 1979).

Kerosene is classified as very slightly toxic based on the lowest oral lethal dose of 28,000 mg/kg in rats (NLM 1987). Kerosene and all other hydrocarbons represent an acute ingestion hazard to humans and, when swallowed, can lead to chemical pneumonia (Doull et al. 1980). Chemical pneumonitis from hydrocarbons, such as kerosene, is described in Doull et al. (1980) as follows:

> An important toxicologic problem associated with the hydrocarbon solvents is the inadvertent or intentional ingestion of gasoline, kerosene, or paint thinners. Although in most instances the acute toxicity of these compounds is quite low, small amounts may be aspirated into the lungs during ingestion, during attempts to induce vomiting, or while pumping the stomach. The response of the lung to small quantities of hydrocarbon solvents is rapid and severe. Relatively small amounts will spread a thin layer over the large moist surfaces of the lung resulting in pneumonitis, pulmonary edema, and hemorrhage.

Kerosene causes moderate local irritation, central nervous system depression, and sometimes mild lesions in the kidneys, liver, bone marrow, and spleen (Gosselin 1976, as cited in NLM 1987). In a 28-day dermal toxicity study with rabbits, kerosene was moderately irritating at the 200- and 1,000-mg/kg/day dose levels and was severely irritating at the 2,000-mg/kg/day dose level (American Petroleum Institute 1983a). Treatment-related skin lesions (acanthotic

dermatitis, hyperkeratosis, and dermal microabscess) and liver lesions (acute multifocal necrosis) occurred at the highest dose (2,000 mg/kg/day). Jet fuel A (a type of kerosene) was mildly irritating to the skin and eyes of rabbits in primary skin and eye studies (Beck et al. 1982). No reactions were observed for guinea pigs used in the same studies (Beck et al. 1982). Rats exposed to 300 mg/m³ for 14 to 75 weeks exhibited morphologic changes and cytoenzymatic changes in the lungs and showed disorders of their acid-base equilibrium (Starek and Kaminski 1981, as cited in NLM 1987).

In a study in which baboons were administered kerosene by various routes, the primate brain appeared to be resistant to direct toxic effects of kerosene (Wolfsdorf 1976). The author hypothesized that the lung and liver are able to filter out sufficient amounts of large doses to protect the brain. Jet fuel A was not reported to be teratogenic in a rat inhalation study at the highest dose tested (400 ppm) (Beliles and Mecler 1982).

Nonthreshold Effects

Diesel oil was nonmutagenic when tested with and without metabolic activation in the Ames assay and in the mouse lymphoma assay. However, it was found to be clastogenic (defined on p. 36) in rat bone marrow cells (Conaway et al. 1982). Kerosene was nonmutagenic when tested with and without metabolic activation in the Ames assay, the mouse lymphoma assay, and the rat bone marrow cell assay (Conaway et. al. 1982). However, because diesel oil and kerosene contain polycyclic aromatic hydrocarbons (PAHs) and other constituents that are known or suspected mutagens, they are considered to be mutagens for this risk assessment.

The oncogenic potential of petroleum fuels is directly related to refinery processing methods used to obtain the petroleum product and the crude oil composition from which the fuel was derived. An evaluation of the composition of petroleum fuels has revealed that a positive correlation exists between PAH content and carcinogenicity in human epidemiology studies or experimental laboratory studies (Bingham et al. 1979).

Diesel fuel is usually a straight-run distillation product composed of a complex variable mixture of hydrocarbons with a boiling point range of 175 °C to 370 °C (DOE 1983) and an aromatic content of 24 percent (Conaway et al. 1982). To date, diesel fuel has not been shown to be carcinogenic. In a 2-year

oncogenic skin painting study, which was terminated after 62 weeks because of the presence of extensive skin lesions, Swiss Eppley mice were exposed to 0.05 mL (41 mg) of diesel fuel products. Skin carcinomas were found in 2 of 50 animals, which was not statistically significant by chi-square analysis (American Petroleum Institute 1983b).

Kerosene is a straight-run distillation product with a boiling point range of 175 °C to 325 °C (NLM 1987) and an aromatic content of 18 percent (Conaway et al. 1982). Higher boiling point (greater than 370 °C) petroleum products that are subjected to additional refinement processes, such as cracking or hydrogenation, and that contain polycyclic aromatic may be carcinogenic to experimental animals (Bingham et al. 1979).

Diesel oil and kerosene contain small amounts of known or suspected carcinogens, including benzo(a)pyrene and benzene (Bingham et al. 1979). Benzo(a)pyrene (BaP), a potent carcinogen, is a PAH that also occurs at low levels in foods and in products of combustion, including cigarette smoke (Bingham et al. 1979). Bioassays indicate that the concentration of this single carcinogen can often serve as a guide in predicting carcinogenic potency, although other substances are also known to be involved (Bingham et al. 1979). There is sufficient evidence to conclude that BaP is carcinogenic in experimental animals: BaP has incited tumors in all of the nine species for which data have been reported, despite the use of different methods of administration (DHHS 1985). These studies reported both local and systemic carcinogenic effects.

For benzene, another aromatic hydrocarbon known to be present in petroleum fuels, there is sufficient evidence to indicate that it is carcinogenic in experimental animals and in humans (DHHS 1985). Benzene has been shown to cause leukemia in chronically exposed workers (DHHS 1985).

Because of the carcinogenicity of the aromatic hydrocarbons found in diesel fuel and kerosene, these light fuel oils are considered carcinogenic for this risk assessment.

The carcinogenic potencies of diesel oil and kerosene have been estimated for this risk assessment based on the potencies of both benzene and BaP. EPA (1980b, as cited in EPA 1984a) has estimated the carcinogenic potency of BaP as 11.5 per (mg/kg/day).

The carcinogenic potency of benzene, however, is much less than that of BaP. EPA (1980a, as cited in EPA 1984b) has estimated the carcinogenic potency of benzene as 0.0445 per (mg/kg/day).

Samples of diesel oil and fuel oil have been found to have a BaP content of only 0.026 ppm, but No. 2 heating oil (which may be subjected to cracking, rather than being a straight-run distillation product) can contain 600 ppb (Bingham et al. 1979). The midpoint of this concentration range (313 ppb) has been used to calculate the carcinogenic potency of diesel oil, although most diesel fuels can be expected to have a lower BaP content. The content of benzene in diesel fuel was assumed to be 28.5 ppm, based on analysis of water extracts of No. 2 fuel oil by Anderson (1975), with corrections for solubility relationships. The resulting estimate of carcinogenic potencies of both diesel oil and kerosene are 0.0000049 per (mg/kg/day). Seventy-four percent of this potency is a result of the BaP component.

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CDFA. California Department of Food and Agriculture.

DHHS. U.S. Department of Health and Human Services.

DOE. U.S. Department of Energy.

EPA. U.S. Environmental Protection Agency.

NLM. National Library of Medicine.

NRC. National Research Council.

USDA. U.S. Department of Agriculture.

USDI. U.S. Department of the Interior.

WHO. World Health Organization.

WSSA. Weed Science Society of America.

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Section E4
Exposure Analysis

Introduction

This section presents the methods used in the herbicide exposure analysis. The terminology of herbicide use and the potential human exposure from that use are discussed in the first subsection.

The second subsection presents the methods used to estimate herbicide doses to members of the public and workers. The methods used for determining lifetime doses to workers and the public to evaluate the risk of cancer are described. The third subsection discusses the results of the routine and accidental dose calculations for the public and workers.

Background Information

Herbicide Characteristics

Most herbicides are packaged and sold by the manufacturer in liquid form as a concentrate with a specified number of pounds of active ingredient, usually between 1 and 10, per gallon of concentrate, and with inert ingredients forming the remaining portion. Many herbicides are also marketed in granular formulations and as wettable powders. Before herbicides are applied, they are mixed with a carrier, usually water, according to the manufacturer's label instructions for the particular treatment purpose and the desired application rate in pounds of active ingredient per acre. The amount of concentrate that produces the desired amount of active ingredient per acre treated is normally mixed with 10 to 15 gallons of carrier for every acre to be treated in aerial applications and with 50 to 100 gallons of carrier for every acre to be treated in ground applications. Herbicide concentrate, stored in 30- to 55-gallon drums, is prepared for application and then is transferred to application equipment by a mixer-loader, who uses a batch truck that has separate storage tanks for the carrier and for the herbicide mixture.

Herbicide application equipment is designed to cover target plants with a minimum of off-target spray movement, called drift. Spray equipment nozzles are designed to produce medium to large droplets because smaller droplets tend to remain airborne and may drift with air currents away from target vegetation. Despite the effectiveness of the application equipment used, some small fraction of the droplets may break up into smaller droplets that the wind could blow offsite. Hand application techniques, such as spot gun and hack-and-squirt, do not use broadcast sprays; thus, these techniques do not produce any appreciable herbicide drift.

Exposure and Dose

Two primary conditions are necessary for a human to receive an herbicide dose that may result in a toxic effect. First, the herbicide must be present in the person's immediate environment—in the air, on the skin, or in food or water—so that it is available for intake. The amount of herbicide present in the person's immediate environment is the exposure level. Second, the herbicide must get into the person's body by some route. If the herbicide is in the air, it may be inhaled into the air passages and lungs. If the herbicide is on the clothing that is in contact with the skin or on the skin itself, it may penetrate the skin. The amount of herbicide that moves into the body by any of these routes constitutes the dose.

While two people may be subjected to the same level of exposure (for example, two workers applying herbicide with backpack sprayers), one may get a much lower dose than the other by wearing protective clothing, using a respirator, or washing immediately after spraying. Exposure, then, is the amount of herbicide available to be taken into the body; dose is the amount of herbicide that actually enters the body.

Worker dose levels were extrapolated from actual field studies that analyzed urine samples from exposed workers. By determining the amount of an herbicide or its metabolites in a worker's urine, it is possible to estimate the exposure (or dose) that the worker has received.

Exposure Analysis Methods

Application Scenarios

To make reasonable estimates of the possible herbicide doses to the public and workers, a

number of application scenarios are used that represent an array of likely treatment situations. Routine application scenarios were designed to provide a range of human dose estimates, from realistic to worst case, for normal operating conditions. Accident scenarios—direct application, spills on the skin, and large spills into bodies of water—are used to estimate the highest doses that could ever be reasonably expected to occur. Actual exposures from all vegetation management projects conducted in the 13 Western States should be within or below the range of doses predicted in these scenarios.

The scenarios specify those characteristics of each kind of herbicide application operation that determine human doses. For example, the number of work hours and the herbicide application rate are used to determine the doses for workers involved in backpack operations. For aerial applications, the number and size of the sites treated in a day's operation are used. To calculate doses to nearby residents who may eat a garden vegetable containing herbicide residue, it is necessary to estimate how much residue is on the vegetable and to specify how much of the vegetable is eaten.

The application scenarios were not intended to show what necessarily *will* happen as a result of a given treatment operation, but what *could* happen if all of the conditions specified in the scenario were met in the actual operations. For example, routine-worst case worker doses are based on actual dose levels found in field exposure studies in which no protective clothing or equipment was worn. If workers were to wear protective clothing and equipment during actual operations, their doses could be significantly lower than the routine-worst case doses estimated here. However, despite all precautions, workers present during treatment operations are likely to be exposed at least to some minimal extent.

Additional factors must be recognized when evaluating the likelihood of a member of the public receiving an herbicide dose. A forest user would receive a dose only in the immediate vicinity of the treatment area and only at the time of the herbicide application. However, because of the limited area of forest being treated and the public's restricted access and use, the possibility of this occurrence is slight. Likewise, a nearby resident would receive an herbicide dose as high as the one estimated in this analysis only if the following conditions were met:

(1) The resident was close enough to a particular treatment area to receive some level of herbicide drift.

(2) The weather conditions on the day of treatment were such that the herbicide happened to drift offsite in the direction of the resident.

A combination of factors makes the possibility of the resident receiving such a dose unlikely. First, most treatment areas are located considerably farther from any residence than the distance assumed in this analysis, which is 600 feet. Second, mitigation measures reduce the likelihood of drift onto a resident, even if one happened to be nearby. Third, there is only a remote possibility that the resident would be present near spraying operations and be unable to avoid spray or not wash herbicide from the skin if sprayed.

Potential Routes of Human Exposure

The potential routes of exposure to humans and nontarget species from herbicide treatment operations are illustrated in Figure E4-1. The routes of exposure considered in this risk assessment in estimating doses to the public that might occur during routine operations or in the event of an accident are listed in Table E4-1 and are described below. Food items and drinking water sources that may lead to ingestion (dietary) exposures and sources of multiple public exposures also are presented.

Potential Human Exposures From Routine Operations

The greatest doses to humans in routine herbicide applications are to workers who may be exposed while (1) mixing herbicide and loading it into application equipment, (2) applying herbicide to vegetation using ground-based equipment, or (3) supervising or monitoring aerial or ground-based herbicide applications. Use of protective clothing and equipment and adherence to proper cleanup procedures and label precautions can lead to significant reductions in the doses to workers.

The most important source of exposure to persons who do not handle the herbicide containers or spray equipment in routine operations is from off-target drift of airborne herbicide spray droplets. Spraying only under favorable weather conditions and using spray equipment that limits the number of smaller spray droplets reduces the amount and extent of drift.
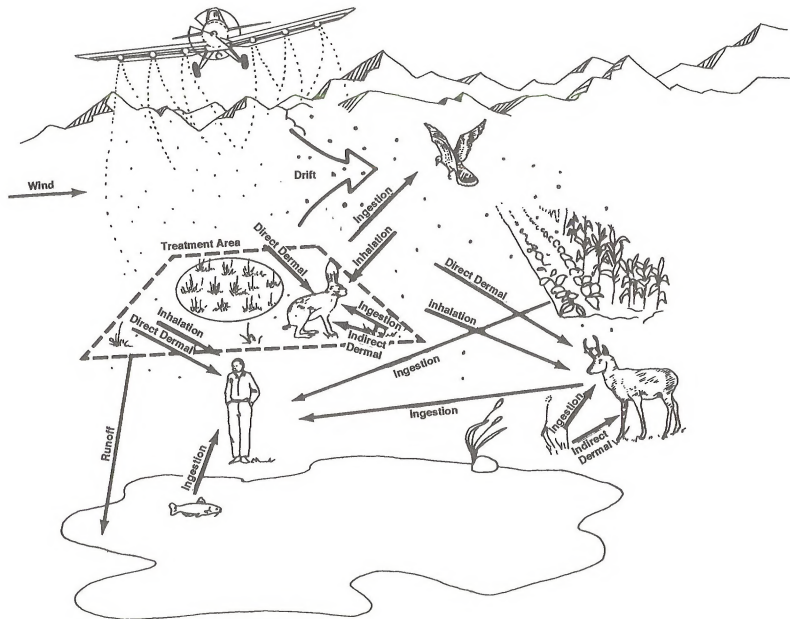
Figure E4-1. Routes of exposure to herbicides in spraying operations.

Table E4-1. Scenarios for Public Exposure Estimation

Exposure	Routes
Spray drift, dermal	Dermal exposure to drift
Vegetation contact, hiker	Dermal exposure to vegetation contaminated with drift
Vegetation contact, picker	Dermal exposure to vegetation contaminated with drift
Drinking water	Oral ingestion of water contaminated with drift
Eating berries	Oral ingestion of berries contaminated with drift
Eating fish	Oral ingestion of fish from water contaminated with drift
Hiker	Dermal exposure to drift + dermal exposure to vegetation contaminated with drift + oral ingestion of water contaminated with drift
Berrypicker	Dermal exposure to drift + dermal exposure to vegetation contaminated with drift + oral ingestion of water contaminated with drift
Angler	Dermal exposure to drift + dermal exposure to vegetation contaminated with drift + oral ingestion of water contaminated with drift + oral ingestion of fish from water contaminated with drift
Nearby resident	Dermal exposure to drift + dermal exposure to vegetation contaminated with drift
Accidents	
Skin spill, concentrate	Dermal exposure from spill of concentrate
Skin spill, mixture	Dermal exposure from spill of mixture
Direct spray, person	Dermal exposure at the full application rate
Drinking directly sprayed water	Oral ingestion of contaminated water
Eating fish from directly sprayed water	Oral ingestion of fish from contaminated water
Immediate reentry, hiker	Dermal exposure to just-sprayed vegetation
Immediate reentry, picker	Dermal exposure to just-sprayed vegetation
Eating directly sprayed berries	Oral ingestion of sprayed berries
Drinking water contaminated by a jettison of mixture	Oral ingestion of contaminated water
Drinking water contaminated by a truck spill	Oral ingestion of contaminated water

During routine operations, workers may be dermally exposed to an herbicide if the herbicide concentrate, mixture, or drifting spray droplets contact their skin or if the herbicide is brushed off of sprayed vegetation. Inhalation exposure may result from breathing without protective devices in the area of the drifting spray droplets or where there are vapors from a volatile herbicide. However, studies have shown that inhalation exposure is small compared with dermal exposure. In this analysis, inhalation doses have not been estimated separately for workers; they are included with dermal doses in the estimated total worker doses based on field experiments measuring herbicide levels in workers' urine.

Field studies of workers have demonstrated that inhalation exposure represents only a small part of the total exposure. Total 2,4-D exposure to truck applicators by way of inhalation, assuming an 8-hour day and a breathing rate of 29 liters per minute, would be a maximum 0.03 milligrams (mg) versus a maximum 18 mg by way of dermal exposure, according to data of Draper and Street (1982). Inhalation, therefore, constituted 0.17 percent of dermal exposure. Nigg and Stamper (1983) calculated inhalation exposure to be 0.03 percent of total body exposure for Florida airboat sprayers. In their study of right-of-way applicators using 2,4-D, 2,4-DP, and picloram, Libich et al. (1984) found dermal exposure to be up to 50 times greater than exposure from inhalation.

Members of the public who are within the area of drift of the smaller spray droplets may also receive dermal and inhalation exposure, but their exposures are relatively low compared to the exposures of workers directly involved in the spraying operations. Herbicide may be ingested by members of the public from food containing herbicide residues. Food items, such as wild berries, may have received some level of herbicide from spray drift. Ingestion exposure could also result from drinking water that has received herbicide drift or from eating fish from a body of water that has received herbicide drift. Because inhalation doses would make a negligible contribution to total doses, only doses from dermal and dietary routes of exposure have been estimated for members of the public.

Potential Human Exposures From Accidents

In the event of an accident, members of the public and workers may be exposed to much greater amounts of herbicide than under normal exposure circumstances. Workers who spill the concentrate or some of the prepared spray mixture on their skin during mixing, loading, or spraying operations, or who are doused if a transfer hose breaks would be dermally exposed. Workers or members of the public who are accidentally sprayed with herbicide because they are beneath a spray aircraft or are too close to a truck or backpack applicator would receive a dermal dose.

The dermal dose would depend on the concentration of herbicide in the spray mix, the area of the sprayed person's exposed skin, the extent to which the person's clothing absorbed herbicide (which depends on the fabric and finish), and the time that elapses before the person can wash. Indirect dermal (reentry) exposure may occur if workers or members of the public brush up against freshly sprayed vegetation in the sprayed area.

Members of the public may accidentally be exposed to the herbicide by eating food or drinking water that has been directly sprayed. For example, members of the public may eat berries that have been directly sprayed. Exposure to even higher levels of herbicide is possible if a container of herbicide concentrate were to break open and spill into a drinking water supply.

Public Doses From Routine Operations

Herbicide doses to the public potentially exposed to routine herbicide applications were estimated using six herbicide application scenarios: 3 routine-realistic and 3 routine-worst case. The hand application scenario was excluded because no drift is involved and the chance that any other type of public contact with the herbicides might occur in these operations is negligible. In the remaining six scenarios, inhalation exposure was not estimated because worker field studies have consistently shown inhalation exposure to be an insignificant fraction of the total herbicide dose received (USDA 1984). Only dermal and dietary routes of exposure were considered in this analysis.

Because no field studies existed on actual doses to the public comparable to those used for estimating worker doses, it was necessary to estimate public doses by modeling the transport and fate of the applied herbicides. Details of the transport and fate modeling are in a following subsection.

Single Routes of Exposure

The following categories of exposure were estimated for all scenarios except public exposures associated with oil and gas sites:

doses resulting from drift, vegetation contact by a hiker or berrypicker, and dietary exposures to water, fish, and wild berries. At oil and gas sites it was assumed that there would be no vegetation contact from berrypicking, dietary exposure from fish, or dietary exposure from water. Dermal exposure from drift and vegetation contact while hiking were the only public exposures assumed to occur there. These scenarios are summarized in Table E4-1.

Multiple Routes of Exposure

In addition to estimating doses to the public from routine operations through the specific exposure routes described above, four categories of persons were assumed to receive cumulative doses through a number of exposure routes: (1) a hiker, (2) a person who picks berries, (3) an angler, and (4) a nearby resident. Each of these persons was assumed to receive an herbicide dose that is the sum of the doses from several routes of exposure as shown in Table E4-1.

It is extremely unlikely that a member of the public will receive simultaneous herbicide doses through more than two of the exposure routes described above. However, to ensure that no possible dose was omitted from the analysis, it was assumed that the hiker would receive dermal exposure from drift as well as vegetation contact from brushing against offsite plants that have received drift. The hiker also would drink water that has received herbicide drift. The berrypicker would receive the same dermal and drinking water exposure from drift as the hiker, but the berrypicker would be exposed to a higher level of vegetation contact exposure from brushing against plants that have received drift because of continuous contact with the berry plants. The berrypicker also would receive exposure from feeding on berries that have herbicide residues from drift.

The angler is assumed to receive the same dermal exposure, vegetation contact, and drinking water exposure as the hiker. Additionally, the angler is assumed to catch and eat fish from a pond that has received herbicide spray drift. The nearby resident would receive the same dermal exposure as the hiker, but does not drink water that has been contaminated with spray drift.

Herbicide Transport and Fate Modeling

The following subsection presents a detailed discussion of the transport and fate modeling used in estimating herbicide doses to the public. Various sources for assumptions and methods of calculation were consulted (Dost 1983, Crump 1983, Simmons 1983, USDA 1984).

Spray Drift. The potential for herbicide sprays to drift onto adjacent lands or into nearby bodies of water was assessed based entirely on the results of studies reported in the scientific literature. The analysis considered deposition on surfaces, including exposed skin, water, game animals, and various classes of plants that may contribute directly or indirectly to the human diet.

Specific field studies were chosen to best represent the equipment and conditions appropriate for each scenario. Unfavorable conditions were chosen to show the degree of drift that could occur under the routine-worst case scenarios. Drift estimates for sprays applied in large range improvement projects were made based on the drift of 2,4-D from a fixed-wing aircraft (Miller 1980). This test was conducted when winds averaged 9.5 mph. Mitigation measures specify no spraying if winds exceed 6 mph, unless the label specifies a lower maximum wind speed. Drift estimates for sprays applied in silvicultural projects were made based on drift of a dye tracer solution sprayed over a coniferous seed orchard by helicopter (Barry et al. 1983). The winds ranged from 4.5 to 9 mph. Drift of sprays applied by ground equipment was estimated based on a field test reported in Yates et al. (1978). In that test, glyphosate was sprayed by a ground sprayer in winds of 8.5 mph.

To facilitate use of the data from these published field tests, a computer program was written to show how residues accumulate from multiple swaths (the long, narrow pattern of herbicide laid down by a broadcast sprayer such as an aircraft) and to correct for various application rates and swath widths. The program was then run to calculate deposition at selected representative distances for a nominal application rate of 1 pound per acre. The results are given in Table E4-2 for each of the three broadcast spray scenarios. The drift calculated for water bodies is intended to represent deposition at the edge of a minimum buffer strip (50 feet for aerial spraying and 20 feet for ground spraying).

Residues on Plants. Herbicide residues on plants on treated sites were estimated based on factors reported by Hoerger and Kenaga (1972). These factors were derived from a large number of studies, and they allow prediction of residues in parts per million (ppm) based on the application rate in pounds

Table E4-2. Offsite Drift Deposition of Herbicides

Application	Deposition (mg/m²) at Distance to Human		Deposition (mg/m²) at Distance to Water and Berries	
	Realistic	Worst Case	Realistic	Worst Case
Aerial	0.0215	1.02	2.000	11.39
Ground Mechanical	0.0462	0.1613	0.0968	0.3500
Backpack	0.0367	0.7289	0.0632	15.000

per acre. These residue estimates were calculated assuming no herbicide degradation, so they apply to conditions immediately after application. Following Hoerger and Kenaga (1972), the plants were classified into broad groups based on vegetative yield, surface-to-mass ratio, and plant interception factors. The residues estimated for each type of plant are intended to represent conservatively realistic estimates.

Offsite plant residues were calculated first for grasses based on the spray drift data discussed in the previous section, using a regression equation that Yates et al. (1978) used to relate spray drift deposition on young wheat plants to that on sampling devices. The deposition was then estimated for other plant groups, including berries and leafy vegetables, by using the same relative factors given by Hoerger and Kenaga (1972), assuming that deposition on young wheat was approximately the same as deposition on range grass.

Residues in Water. Residues in water were calculated assuming that the water is 4 feet deep and that the herbicide spray drifts directly downwind to the water body over a minimum drift distance. The drift deposition is detailed in Table E4-2. The concentration in water is calculated as follows:

$$CONC = DRIFT \times 1/4 \text{ feet} \times 3.28 \text{ feet/m} \times 0.001 \text{ m}^3/L$$

where:

$CONC$ = Herbicide concentration in mg/L
$DRIFT$ = drift deposition in mg/ft² (from Table E4-2).

The actual residues in water would be less under more favorable spray conditions, at greater distances, or with deeper water bodies. For example, if the water were 8 feet deep, the residues would be only one-half of those

calculated for this analysis. Dilution or degradation would also decrease residues.

Residues in Fish. Residues in fish were calculated assuming that the fish lived in and were caught in water 4 feet deep, directly downwind of a treated site, with the drift distances given in Table E4-3. For most of the herbicides considered in this analysis, where the bioconcentration factor was unknown, the concentrations in fish were conservatively taken to be 25 times the particular herbicide's concentrations in water. For the herbicides for which a bioconcentration factor is known—atrazine, diuron, and tebuthiuron—the literature bioconcentration factor was used. A bioconcentration factor of 10 was used for tebuthiuron, a value of 5 was used for atrazine, and a bioconcentration factor of 20 was used for diuron (Koeman et al. 1969).

Dermal Doses to the Public

Dermal doses from drift were estimated by assuming that 2 square feet of skin are exposed (Dost 1983) and the level of deposition on skin is the same as that found on the sampling sheets used in the drift monitoring studies. The dose was calculated as the deposited amount multiplied by the dermal penetration rate, then divided by body weight (50 kg).

Indirect dermal exposure resulting from contact with foliage with surface residues of drifted herbicide was calculated by using the "unified field model" of Popendorf and Leffingwell (1982) and Popendorf (1985). This model was developed to estimate the possible doses and effects of insecticides on agricultural workers; however, it was only used to estimate exposure for this analysis. The model was applied to estimate the relatively heavy exposures that could result from extensive foliage contact, such as that which would be

Table E4-3. Drift Distances Used in Exposure Estimations

| | | Drift Distances (feet) | | | |
| | | To Water and Berries | | To Humans | |
Program	Scenario	Realistic	Worst Case	Realistic	Worst Case
Rangeland	Aerial	200	100	600	200
	Backpack	20	10	50	20
	Ground Mechanical	50	25	100	50
Public-Domain Forest Land	Aerial	200	100	600	200
	Backpack	20	10	50	20
	Ground Mechanical	50	25	100	50
Oil and Gas Sites	Aerial	200	100	600	200
	Backpack	20	10	50	20
	Ground Mechanical	50	25	100	50
Rights-of-Way	Aerial	200	100	600	200
	Backpack	20	10	50	20
	Ground Mechanical	50	25	100	50
Recreation and Cultural Sites	Aerial[a]	—	—	—	—
	Backpack	20	10	50	20
	Ground Mechanical	50	25	100	50

[a]Aerial applications are not used on BLM-managed recreation and cultural sites.

experienced in berrypicking. The model takes into account the following:

(1) The residue on foliage at any point in time after application (this analysis assumes no decay after initial application)

(2) A crop-specific residue transfer coefficient [square centimeters per hour (cm²/hour)]

(3) The exposure period in hours

(4) The dermal penetration rate for each herbicide and the body mass of a human (50 kg)

The residue transfer coefficient has been determined for a few agricultural situations.

The value of 1,600 cm²/hour for this coefficient was used in this analysis to estimate doses to berrypickers. This value, derived from data collected for grape harvesting (Popendorf 1985), represents a relatively high exposure situation. People engaged in activities involving less foliage contact, for example, tree planting, can be expected to receive doses that are considerably less.

Dermal doses resulting from incidental contact with foliage, represented in the scenarios by vegetation contact for the hiker, were estimated by another method. Lavy et al. (1980) measured the level of 2,4,5-T on cloth patch samplers attached to a person who walked through a treated forest area. The residues were less than the detection limit of 0.01 mg per 100 cm² patch, but in this analysis a conservative assumption was made

that the residues were at the detection limit. The area of clothing contacting foliage was assumed to be 40 percent of the total human surface area, and 10 percent of the total area was assumed to be bare skin contacting foliage. The same dermal penetration rates discussed previously were applied to bare skin, but the penetration through clothing was assumed to be 30 percent over a 6-hour period, based on work by Newton and Norris (1981).

Dietary Doses to the Public

Drinking Water. Herbicide doses to individuals were calculated assuming they drink 2 liters (L) of water per day, as follows:

$$DOSE = CONC \times AMT \times (1/BWT)$$

where:

DOSE = Dietary dose due to drinking contaminated water (mg/kg/day)

CONC = Concentration of herbicide in water (mg/L)

AMT = Water consumption (2 L/day)

BWT = Body weight (50 kg)

Eating Berries. Calculations for public dietary doses as a result of eating contaminated berries are based on the following equation:

$$DOSE = RESIDUE \times AMT \times (1/BWT)$$

where:

DOSE = milligrams of herbicide consumed per kilogram of body weight (mg/kg)

RESIDUE = mg herbicide per gram food

AMT = amount of diet item consumed (400 g)

BWT = body weight of person; assumed to be 50 kg

Eating Fish. To illustrate the method used to determine human dietary exposures from consuming fish contaminated with herbicides, the following example is provided for a routine-realistic aerial operation on forests using atrazine:

The first step is to calculate the herbicide concentration per pound applied in a water body 4 feet deep. According to Table E4-2,

the value of drift to water from realistic aerial applications is 2.000 mg/m^3. The concentration of herbicide in a pond 4 feet deep per pound applied per acre is:

$$\frac{2.000 \text{ mg}}{m^3} \times \frac{1}{4 \text{ ft}} \times \frac{ft}{0.305 \text{ m}}$$
$$\times \frac{0.001 \text{ m}^3}{L} = 1.64 \times 10^{-3} \text{mg/L}$$

The concentration of atrazine is adjusted for the 4-lb/acre application rate on forests:

$$0.00164 \text{ mg/L} \times 4.00 = 0.00656 \text{ mg/L}$$

The concentration in the fish is based on the bioconcentration factor of atrazine. This assumes that a fish accumulates 5 mg of atrazine per kilogram body weight for every 1 mg in water and is calculated as follows:

$$0.00656 \text{ mg/L} \times 5 \text{ mg/kg} = 0.0328 \text{ mg/kg}$$

The dose to a 50-kg human based on consumption of 0.4 kg of fish is:

$$\frac{(0.0328 \text{ mg/kg} \times 0.4 \text{ kg})}{50 \text{ kg}} = 0.00026 \text{ mg/kg}$$

Workers' Doses From Routine Operations

Herbicide doses to workers involved in routine operations were estimated using 8 herbicide application scenarios: 4 routine-realistic and 4 routine-worst case scenarios. For each application scenario, worker categories were chosen to represent the normal range of work activities in terms of potential herbicide exposure. These are summarized in Table E4-4. Other categories of workers may experience less exposure, but no category of workers in the field is expected to experience greater exposure than the types of workers considered in this analysis.

Worker Categories and Calculations in Routine Operations

Worker dose levels were extrapolated directly from worker doses determined by urine analysis in field studies of actual herbicide treatment operations. Because the field studies showed what dose levels are experienced in actual operations, they were considered the most appropriate basis for estimating the doses to BLM herbicide applicators involved in the same or similar vegetation management practices. Those studies are discussed in the next subsection.

Table E4-4. Worker Exposure Categories

Application	Workers
Aerial	Pilot
	Mixer-loader
	Fuel truck operator
Backpack	Applicator
Ground Mechanical	Applicator
	Mixer-loader
	Applicator/mixer-loader
Hand	Applicator

Dose estimates were scaled to the anticipated work hours and herbicide application rates specified in each of 8 application scenarios.

Routine-realistic. To estimate routine-realistic worker doses, the analysis used average dose levels found by urine analysis in field studies of workers exposed while spraying 2,4-D using the same application method. Nominal dose levels in milligrams of herbicide received per kilogram of body weight (mg/kg) for workers in each category were derived from these average dose levels by dividing by the field study acreage and application rate.

Typical application rates for the routine dose scenarios are listed in Table E4-5.

Doses for each worker category were estimated by extrapolating from the average dose levels found in field studies of workers exposed to 2,4-D using the same application method. The following steps were involved:

(1) The average dose observed in the 2,4-D field study was expressed in terms of dose (mg/kg) per pound of active ingredient applied.

(2) The typical number of acres treated was multiplied by the herbicide's typical application rate to determine the number of pounds of active ingredient used in the scenario.

(3) The herbicide-specific dose was determined by multiplying the pounds of herbicide applied by the dose of 2,4-D per pound of 2,4-D applied for that

worker category in the field studies and then adjusting for the particular herbicide's dermal penetration rate. The dermal penetration rates used in the analysis were 6 percent for 2,4-D (Feldman and Maibach 1974), 0.48 percent for picloram (Lavy et al. 1984), 5 percent for dicamba (Draper and Street 1982), 0.1 percent for amitrole, and 10 percent for the other eighteen herbicides and carriers (USDA 1984).

The aerial, ground mechanical, and backpack worker doses were calculated for each herbicide and each scenario using the following equation:

$$DOSE = STDOSE \times RATE \times ACRES \times DPRCF$$

where:

DOSE = dose to worker in mg/kg

STDOSE = dose from field study with 2,4-D (reported in Table E4-6)

RATE = application rate for the herbicide from Table E4-5

ACRES = the number of acres treated in 1 day for the scenarios from Table E4-7

DPRCF = the dermal penetration rate correction factor derived from the ratio of the dermal penetration rate of the herbicide to the dermal penetration rate of 2,4-D (6 percent)

An example calculation using this equation follows for a pilot's dose of amitrole under the routine-realistic aerial rangeland scenario:

7.55×10^{-5} mg/kg/lb applied
$\times 2.0$ lb/acre $\times 320$ acres/day
$\times (0.1/6) = 0.00081$ mg/kg/day

Worker doses for hand application methods were calculated using the following equation:

$$DOSE = (STDOSE/STHRS) \times (LBGAL/STLBGAL) \times HRS \times DPRCF$$

DOSE = dose to worker in mg/kg

STDOSE = dose from field study with 2,4-D

STHRS = number of hours worker in study applied 2,4-D

Table E4-5. Typical Herbicide Application Rates for BLM Vegetation Treatment Programs

Herbicide	Application Rate (pounds active ingredient/acre)				
	Rangeland	Public-Domain Forest Land	Oil and Gas Sites	Rights-of-Way	Recreation and Cultural Sites
Amitrole	2	2	4	2	—
Atrazine	1	4	10	4	1
Bromacil	—	—	8	8	—
Chlorsulfuron	—	0.125	0.141	0.141	0.125
Clopyralid	0.5	—	12	12	—
2,4-D	4	4	4	4	3
Dalapon	3	4	4	4	4
Dicamba	4	4	8	4	4
Diuron	—	—	10	4	—
Glyphosate	4	2	4	4	4
Hexazinone	0.67	2	4	2	2
Imazapyr	1	1.5	1.5	1.5	1.5
Mefluidide	—	—	0.25	0.25	—
Metsulfuron methyl	—	—	0.075	0.075	—
Picloram	2	2	3	3	2
Simazine	—	4	10	4	1
Sulfometuron methyl	—	—	0.563	0.563	—
Tebuthiuron	0.5	1.5	8	1.5	0.5
Triclopyr	1.5	2	4	4	1.5
Carriers					
Diesel oil	2	2	2	2	2
Kerosene	2	2	2	2	2

Table E4-6. Doses From Worker Exposure Studies Used to Calculate Doses to BLM Workers

| Application | Worker | Dose (mg/kg per pound active ingredient applied) | |
		Realistic	Worst Case
Aerial	Pilot	7.55×10^{-5}	2.511×10^{-4}
	Mixer-loader	1.08×10^{-4}	3.201×10^{-4}
	Fuel truck operator	2.43×10^{-6}	7.75×10^{-6}
Backpack	Applicator	7.5639×10^{-2}	1.895×10^{-1}
Ground Mechanical	Applicator	1.71×10^{-4}	7.95×10^{-4}
	Mixer-loader	1.74×10^{-4}	3.30×10^{-4}
	Applicator/mixer-loader	2.40×10^{-4}	4.46×10^{-4}
Hand	Applicator	2.85×10^{-2}	4.78×10^{-2}

Table E4-7. Acres Treated per Day for Worker Exposure Scenarios

Program	Scenario	Acres Treated	
		Realistic	Worst Case
Rangeland	Aerial	320	640
	Backpack	2	4
	Ground Mechanical	60	100
	Hand[a]	—	—
Public-Domain Forest Land	Aerial	50	200
	Backpack	2	4
	Ground Mechanical	25	100
	Hand	2	4
Oil and Gas Sites	Aerial	50	100
	Backpack	2	4
	Ground Mechanical	20	50
	Hand	2	4
Rights-of-Way	Aerial	50	300
	Backpack	2	4
	Ground Mechanical	25	50
	Hand	2	4
Recreation and Cultural Sites	Aerial[b]	—	—
	Backpack	2	4
	Ground Mechanical	10	20
	Hand	2	4

[a]Hand applications are not used on BLM-managed rangeland.
[b]Aerial applications are not used on BLM-managed recreation and cultural sites.

LBGAL = pounds of active ingredient per gallon of herbicide (Table E4-8)

STLBGAL = pounds of 2-4,D applied by workers in study

HRS = number of hours per day worker applies herbicide in scenario

DPRCF = the dermal pentration rate correction factor derived from the ratio of the dermal penetration rate of the herbicide to the dermal pentration rate of 2,4-D (6 percent)

An example calculation using the equation for a worker applying amitrole by the hack-and-squirt method in the routine realistic scenario is as follows:

$$DOSE = (0.0285 \text{ mg/kg/day/6 hours})$$
$$\times (2 \text{ lb/gal})/(3.34 \text{ lb 2,4-D/gal})$$
$$\times 6.4 \text{ hours/day} \times (0.1/6)$$
$$= 0.0003 \text{ mg/kg/day}$$

Routine-Worst Case. Routine-worst case worker doses were estimated for the same worker categories used in the routine-realistic scenarios. However, the site size, application rate, equipment type, meteorological conditions, and duration of exposure used were those that would lead to the highest levels of exposure in herbicide treatment operations. Herbicide-specific dose levels in the routine-worst case scenarios were again derived from the worker field studies and weighted for application rate and hours exposed, but here the 97.5 or 95 percentile of the field study doses were used for extrapolating to the nominal dose in mg/kg/hour for a 1-lb/acre application rate. Application rates used in the routine-worst case scenarios are listed in Table E4-9.

Field Studies of Worker Exposure to 2,4-D

Field studies of the exposures and resultant doses of workers using a variety of application equipment have been conducted on 2,4-D by Lavy et al. (1982), Lavy et al. (1984), Lavy et al. (1987), Nash et al. (1982), and Franklin et al. (1982). Lavy et al. (1982) monitored three helicopter spray crews for worker exposure to 2,4-D using portable air filters, denim patches, and urine analysis on two separate spraying dates; the first observing normal precautions, the second using special protective clothing and procedures. Nash et al. (1982) monitored exposure of workers to 2,4-D during aerial spraying in Washington and ground spraying in

North Dakota under normal spray conditions (that is, without special precautions).

Lavy et al. (1984) investigated herbicide exposure to four spraying crews of 20 workers each, monitoring urine levels over two 5-day periods. In Lavy et al. (1987), similiar studies were performed on forestry ground workers using backpack, injection bar, hypohatchet, and hack-and-squirt equipment to control unwanted vegetation.

Franklin et al. (1982) estimated worker exposure in pasture brush clearing operations in Saskatchewan using techniques similar to Lavy et al. Urine samples were collected from personnel who conducted operations on 3 of 4 consecutive days.

All of the typical doses extrapolated from the worker studies above are based on work crews wearing protective clothing and taking special precautions against exposure. Doses for extreme worker exposure assumed that no protective clothing was worn and no special precautions were taken. Doses from the worker studies that were used to calculate exposures are in Table E4-6.

Effects of the Use of Protective Clothing

The use of protective clothing can substantially reduce worker doses, as shown in field studies of worker exposure, and thereby increase their margins of safety. Protective clothing can reduce worker exposures by 27 to 99 percent, as shown in a number of relevant field studies. Realistic exposures were computed assuming protective clothing is worn. The calculated worst case doses were based on the assumption that workers work with bare hands and wear ordinary work clothing, such as cotton pants and short-sleeve shirts. BLM requires employees applying herbicides to wear clothing that affords more protection. Typical protective clothing often includes long-sleeve shirts or coveralls, gloves, and hats.

Research has shown that such protective clothing can substantially reduce worker exposure. For example, in right-of-way spraying, doses received by spray gun applicators wearing clean coveralls and gloves were reduced by 68 percent compared to doses without this protection (Libich et al. 1984). During an aerial spraying operation, mixer-loaders wearing protective clothing reduced their exposure by 27 percent and other crew members reduced their exposure by 58 percent compared to the levels observed without precautions (Lavy et al. 1982).

Table E4-8. Maximum Herbicide Concentrations in Concentrate, Drums, and Batch Trucks

Herbicide	Herbicide Content (pounds active ingredient)		
	Gallon Concentrate	50-Gallon Drum	2,000-Gallon Batch Truck
Amitrole	2	100	800
Atrazine	4	200	800
Bromacil	4	200	400
Chlorsulfuron	4	200	4,000
Clopyralid	4	200	4,000
2,4-D	4	200	800
Dalapon	—[a]	—	300
Dicamba	4	200	800
Diuron	4	200	640
Glyphosate	3	150	1,000
Hexazinone	2	100	600
Imazapyr	4	200	4,000
Mefluidide	4	200	4,000
Metsulfuron methyl	4	200	4,000
Picloram	2	100	1,000
Simazine	4	200	1,000
Sulfometuron methyl	4	200	4,000
Tebuthiuron	—[a]	—	1,200
Triclopyr	4	200	1,600
Carriers			
Diesel oil	2	100	1,000
Kerosene	2	100	1,000

[a]BLM does not purchase liquid formulations of dalapon and tebuthiuron.

Table E4-9. Maximum Herbicide Application Rates for BLM Vegetation Treatment Programs

Herbicide	Application Rate (pounds active ingredient/acre)				
	Rangeland	Public-Domain Forest Land	Oil and Gas Sites	Rights-of-Way	Recreation and Cultural Sites
Amitrole	2	2	9.9	9.9	—
Atrazine	1	4	40	40	1
Bromacil	—	—	16	16	—
Chlorsulfuron	—	0.125	0.141	0.141	0.125
Clopyralid	0.5	—	12	12	—
2,4-D	6	8	4	4	3
Dalapon	3	4	22	22	4
Dicamba	8	4	8	8	8
Diuron	—	—	32	32	—
Glyphosate	5	3	4	4	5
Hexazinone	0.67	3	10.8	10.8	3
Imazapyr	1	1.5	1.5	1.5	1.5
Mefluidide	—	—	0.25	0.25	—
Metsulfuron methyl	—	—	0.075	0.075	—
Picloram	2	2	3	3	2
Simazine	—	4	40	40	4
Sulfometuron methyl	—	—	0.563	0.563	—
Tebuthiuron	4	5	16	16	4
Triclopyr	1.5	4	8	8	1.5
Carriers					
Diesel oil	2	2	2	2	2
Kerosene	2	2	2	2	2

During insecticide applications to orchards, mixers reduced their exposure by 35 percent and sprayers reduced their exposure by 49 percent by wearing coveralls (Davies et al. 1982). Putnam and co-workers found that nitrofen applicators and mixer-loaders wearing protective clothing reduced their exposure by 94 to 99 percent compared to the doses experienced without protection (Waldron 1985). Although protective clothing generally reduces worker exposure and resulting doses, the degree of protection depends on the application system, the work practices, and the specific herbicide. In one case, workers wearing protective clothing did not receive significantly lower doses than workers with less clothing (Lavy et al. 1984). In this case, backpack applicators had to treat and move through dense vegetation that was taller than themselves.

Most exposure to herbicide applicators is dermal, not inhalation (Kolmodin-Hedman et al. 1983), so the use of respirators is only minimally effective in reducing exposure. The hands are the site of the greatest potential herbicide exposure, and rubber gloves are generally quite effective in preventing exposure to hands (Putnam et al. 1983).

Based on a review of the field studies, protective clothing was found to reduce worker doses by the following amounts:

Type of Worker	Percentage Reduction in Dose
(1) Aerial application pilot	57.1
(2) Aerial application mixer-loader	27.1
(3) Backpack applicators	68.8
(4) Mechanical ground applicator	68.1
(5) Mechanical ground mixer-loader	27.1
(6) Mechanical ground mixer-loader/ applicator	48.4
(7) Hack-and-squirt and spot gun applicators	57.6

Estimation of Doses to Workers and the Public From Accidents

The following scenarios were used to estimate the doses that would result from exposure to high amounts of herbicide that could occur in accidents.

Accidental Spraying

In this scenario a person is accidentally sprayed with herbicide because they are beneath a spray aircraft. Contact with just-sprayed vegetation resulting in dermal doses to hikers and berry-pickers is also evaluated.

To calculate the dose to a person directly sprayed at the full per-acre application rate, the maximum application rates shown in Table E4-9 were converted to milligrams per square feet (mg/ft^2). It is assumed that 2 square feet of human skin are exposed (Dost 1983).

For example, the maximum application rate of amitrole in any scenario is 9.9 pounds of active ingredient per acre (lb a.i./acre). This can be converted to mg/ft^2:

9.9 lb a.i./acre x 453,600 mg/lb x acre/43,539 ft^2 = 103.1 mg/ft^2

If 2 square feet of skin were exposed on a 50 kg person, the surface deposit would be 206.2 mg.

The absorbed dose must consider the dermal penetration rate: 206.2 mg x 0.001 (dermal penetration rate) = 0.206 mg absorbed. This is equivalent to a dose of 0.004 mg/kg.

Reentry exposure to the public is estimated assuming a hiker walks through a treated area after an operation has been completed, even though the area is posted. Reentry exposure is also calculated for an individual who picks berries for 4 hours in a treated area, even though the area is posted.

Accidental dietary exposure is derived by assuming that an individual eats berries that have been directly sprayed, rather than food items receiving only spray drift, or fish taken from directly sprayed water bodies, or who drinks water from those water bodies.

Spills

Members of the public may receive herbicide exposure by way of drinking water if a load of herbicide mixture is spilled or if a container of herbicide concentrate breaks open and spills into a drinking water supply. Workers may

spill concentrate or prepared spray mixture on their skin during mixing, loading, or spraying operations, or be doused when a transfer hose breaks.

Accidental ingestion doses were estimated by modeling the dilution of herbicide concentrate or mixture in a body of water of a given size. Accidental dermal doses were derived from modeling the dermal penetration of herbicide concentrate or mixture for direct exposures.

An individual receives an accidental ingestion exposure resulting from a major spill by drinking water from a pond that has been contaminated by a dump of 80 gallons of herbicide mix as from a helicopter, or 2,000 gallons of spray mix from a batch truck. Two thousand gallons is approximately the largest amount of spray mix that might be carried by a tank truck supplying a large aerial spraying operation. Eighty gallons is approximately the largest load that can be carried by the types of helicopters used in the 13 Western States. The maximum herbicide concentrations in drums and batch trucks are shown in Table E4-8. The pond is assumed to be 0.25 acre in area and 4 feet deep and to have no inflow or outflow. A person is assumed to drink 2 liters of water after complete mixing has occurred.

Direct dermal exposures were calculated for spills of 0.5 liter of herbicide concentrate (if liquid concentrates are used) or 0.5 liter of the most concentrated spray mixture. The person exposed during the spill is assumed to weigh 50 kilograms, and most of this person's surface area (1.36 m^2 or 14.6 ft^2) is thoroughly wetted by the solution. Denim fabric commonly used in clothing retains about 57.5 milliliters of solution per square foot (Weeks 1985), and absorption of herbicide through the cloth was calculated as before, based on Newton and Norris (1981). However, 20 percent of the solution was assumed to wet bare skin. A spill resulting in this exposure could result from broken hoses, spilled containers, or emergency and accidental dumps by helicopters.

Estimation of Lifetime Doses to the Public and Workers

Lifetime exposures to the public for 2,4-D, picloram, amitrole, atrazine, asulam, bromacil, glyphosate, simazine, kerosene, and diesel oil were derived by assuming that a realistic estimate would be four exposures per year for 5 years. The average exposure was assumed to be a sum of 95 percent of the realistic dose and 5 percent of the worst case dose.

Doses used in the cancer risk analysis for the ten chemicals were derived by combining available information on the number of days per year an individual worker may spray an herbicide using a particular application method and estimates of the expected daily dose and the number of years of employment. Two expected daily doses were calculated based on the worst case dose and the realistic dose in all routine scenarios. The realistic cases assume that workers are employed in pesticide application for 10 years.

The expected daily dose for a worker was assumed to be equal to the sum of 90 percent of the realistic dose and 10 percent of the worst case dose. Average numbers of exposures per lifetime were used with expected daily doses for each scenario to derive lifetime doses. Lifetime doses were derived by multiplying expected daily dose levels estimated in worker scenarios by estimates of the highest number of days a worker is likely to be engaged in the particular type of application method. Exposures per lifetime in the realistic scenarios were estimated to be the following: aerial, 60; right-of-way, 90; backpack, 100; and hand application, 140.

Effect of Body Size on Exposure

All doses estimated in the exposure analysis were calculated for a representative 50-kg person. This weight was chosen to represent an adult of less than average weight, so that doses to adults would be calculated in a conservative manner. Doses for a larger person would be less in terms of milligrams per kilograms (mg/kg) of body weight. For example, a 70-kg person would receive approximately 25 percent more herbicide than a 50-kg person by dermal exposure because of that person's greater surface area. A 70-kg person would also receive an average of about 25 percent more herbicide by dietary exposure routes because both metabolic rate and dietary intake are related to body surface area, which is approximately proportional to body weight raised to the 2/3 power:

$$\frac{(70)^{2/3}}{(50)^{2/3}} = 1.25$$

However, a 70-kg person also has a body weight greater than a 50-kg person, by a greater factor:

$$\frac{70}{50} = 1.4$$

The combined effect of these two factors is that a 70-kg person will receive a dose In mg/kg that is only 89 percent as great as for a 50-kg person.

Conversely, smaller people can be expected to receive greater doses In terms of mg/kg body weight. A 20-kg child will receive only about 54 percent as much herbicide as a 50-kg person, but the child's weight is only 40 percent as great. The net effect Is that a 20-kg child will receive a dose that is approximately 35 percent greater In terms of mg/kg than it would be for a 50-kg person.

Exposure Analysis Results

This subsection discusses the results of the exposure analysis. Doses to the public and workers estimated for routine operations and for accidents are summarized here.

Doses to the Public

In comparing the doses received by the public or workers, the reader must be cautioned that even though one herbicide may be received at a higher dose than another herbicide, it does not necessarily mean that the expectation of injury is greater. Chemical toxicity is a function of the dose of chemical received and the particular toxicological properties of the chemical in question. For example, while chemical A may be received at a dose 10 times higher than chemical B, chemical A may be 100 times less toxic than chemical B and therefore present less possibility of injury.

In a broad overview, the herbicide doses to the public are generally much lower than those received by the workers, as would be expected. Also, as would be expected from the terms used in this exposure assessment, herbicide doses that are received from worst case situations are greater than herbicide doses received during realistic exposures. Accidental doses are much greater than worst case doses.

The scenario that presents the lowest doses to the public is the one involving recreational and cultural site exposures. This is because lower herbicide application rates are used on these sites than on other sites. Highest public exposures to herbicides are found on the oil and gas sites, as well as the rights-of-way; the highest herbicide application rates can be found on these sites.

The doses to a member of the public who should happen to walk through a recently sprayed area are extremely small. The greatest possible dose to a person would be expected from the worst case exposure in an oil and gas or right-of-way site because these are the sites where the highest application rate is used.

Depending upon the chemical and scenario in question, the estimated doses resulting from vegetation contact for a berrypicker or the estimated doses from consuming contaminated fish are generally the highest doses. In descending order, doses resulting from drinking contaminated water are generally followed by eating contaminated berries, and these are both followed by dermal exposure to herbicides due to spray drift.

Herbicide doses resulting from aerial spraying are generally higher than doses received from either backpack or mechanical ground application of herbicides. This is a function of the greater expected herbicide drift from aerial application. The dose received is also linked to the assumed buffer strip between application area and receptor. While the drift from aerial spraying is greater than other application methods, the incorporation of larger buffer strips during aerial applications helps to reduce the impact of drift from these operations.

Herbicide doses that are received through multiple exposures are greater than those doses that are received individually. The berrypicker usually receives the greatest dose from multiple herbicide exposures. However, depending upon the particular herbicide and scenario, the angler may receive a higher dose.

Doses to Workers

As was previously stated, the herbicide doses received by workers are generally greater than those received by the public. Also, the doses received under worst case scenarios are greater than those doses received under realistic scenarios.

In routine-realistic aerial application situations, mixer-loaders will receive the greatest herbicide exposure. This is because they have more contact with herbicide concentrates and mixtures than other aerial crew members. The mixer-loader exposures are less than those of the pilot exposures. Fuel truck operators are predicted to have the least exposure to herbicides. Under the routine-worst case aerial application scenarios, the relative doses are the same: mixer-loaders > pilots > fuel truck operators.

Predicted doses are generally greatest for those scenarios in which larger numbers of acres may be sprayed, such as rangeland or forests. However, depending upon the particular herbicide, an aerial crew member's predicted dose may be greater under the right-of-way or oil and gas sites scenarios; even though the acreage sprayed in these situations is much less, the application rate is higher and may outweigh the relative difference in acreage sprayed.

The doses that mechanical ground application crews receive under routine-realistic scenarios are generally lower than the doses aerial crews receive. Mechanical ground crew applicators and mixer-loaders receive approximately the same herbicide dose, while a person who performs the role of applicator/mixer-loader is predicted to receive greater herbicide doses than either a mixer-loader or applicator. In the worst case scenarios, the applicator doses are greater than both the mixer-loader and the applicator/mixer-loader, and the mixer-loader will receive the smallest dose of the three types of crew members. Generally speaking, the doses received by the ground crew members under worst case conditions will be greater than the aerial crew under worst case doses.

Backpack application crew members will receive herbicide doses similar to those of the mechanical ground application crew members, under routine realistic situations. Under worst case considerations, the backpack herbicide applicators will often receive approximately 10 times less herbicide dose than the least exposed ground crew member, the mixer-loader.

Hand application crews generally receive herbicide doses that are similar to, but slightly greater than, aerial crew members under routine-realistic scenarios. Under worst case circumstances, the doses these workers may receive is likely to be increased by a factor of approximately three. This increase is much less than the increase potentially experienced by other application crew members, which may be a factor of 10 or more in many cases.

Doses From Accidental Exposures to Herbicides

The accidental herbicide exposures that are presented in this exposure assessment predict that significant herbicide doses could be received in such events. In many cases, the accidental herbicide doses the public receives are similar to the doses workers receive under their assumed worst case situations. An accidental spill of an herbicide concentrate onto a worker's clothing and skin produces the largest estimated dose resulting from an accident. The smallest calculated accidental dose comes from the accidental entry by a person into an area that has just received an herbicide spray; this dose is similar to the doses aerial crew members typically receive under routine realistic situations. Accidental herbicide doses, except those associated with the consumption of herbicide from water, are similar to the doses that a worker might receive in many instances.

The accidental consumption of contaminated water can lead to high herbicide doses. Since the herbicide concentration is the same in the assumed helicopter jettison into a reservoir and a batch truck spill into a similar reservoir, the difference in the potential dose is caused by the much larger volume of herbicide the truck could potentially spill, 2,000 gallons versus 80 gallons by the helicopter.

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Section E5
Risk Analysis

This section analyzes the risks to the health of members of the public and workers that may result from exposure to any of the herbicides or carriers proposed for use on BLM-managed lands in the 13 Western States. The analysis uses the human exposure levels estimated in Section E4 and the laboratory-determined toxicity reference levels described in Section E3.

The first subsection describes the methods used to evaluate human health risks, including the risks of acute toxic effects, chronic systemic effects, effects on reproduction (maternal and fetal toxicity and birth defects), and cancer. The second subsection presents the results of the risk analysis for each of the programs for the herbicides and carriers. The third subsection discusses the risks of other toxic effects including mutagenicity, synergistic effects, effects on sensitive individuals, and cumulative effects. The final subsection discusses the use of protective clothing.

Methodology for Assessing Health Risks

Risks of Acute, General Systemic, and Reproductive Effects

In this risk analysis, the risks to humans exposed to the 19 herbicides and 2 carriers were quantified by comparing the representative doses estimated in Section E4 with the results of toxicity tests on laboratory animals described in Section E3. To quantify the risks of threshold effects, the doses estimated for exposed individuals were compared to laboratory no-observed-effect levels (NOELs) determined in the most sensitive animal test species. The ratio between the animal NOEL and the estimated human dose is referred to in this analysis as the margin of safety (MOS):

$$MOS = NOEL/dose$$

The margin of safety is used to account for the uncertainty inherent in relating doses and effects seen in animals to estimated doses and effects that might be experienced by humans. For example, an MOS of 100 means that the laboratory-determined no-observed-effect level is 100 times the estimated human dose; an MOS of 10 means the laboratory-determined no-effect level is 10 times the estimated human dose.

The larger the MOS (that is, the smaller the estimated human dose compared to the animal NOEL), the lower the presumed risk to human health. As the estimated dose to humans approaches the animal NOEL, the presumed risk to humans increases. When an estimated dose exceeds a NOEL, the ratio is reversed (the dose is divided by the NOEL) to indicate the factor by which the estimated dose exceeds the laboratory no-observed-effect level. In this case a minus sign is attached to the MOS to indicate that the estimated dose exceeds the NOEL. An MOS of -5, for example, means that the estimated dose is 5 times the laboratory-determined NOEL.

A negative MOS indicates that the estimated dose (given all the assumptions of the exposure situation) may produce some toxic effects, although it must be remembered that the MOS is based on a laboratory dose level that produced no toxic effects in test animals. When repeated doses to humans are much higher than the animal NOEL, there is a clear risk of harmful effects. Conversely, when the human dose is small compared with the animal NOEL (for example, resulting in an MOS greater than 100), the risk to humans can be judged low or negligible.

All of the NOELs used in this risk analysis are based on (or take into account) long-term exposure. A dose estimate that exceeds the laboratory test animal NOEL does not necessarily lead to the conclusion that there will be toxic effects. As an estimated dose approaches or exceeds an animal NOEL, the risk of toxic effects greatly increases, but comparing one-time or once-a-year doses (such as those experienced by the public or in an accident) to NOELs derived from repeated doses in lifetime studies tends to exaggerate the risk from those rare events.

Systemic effects are evaluated based on the lowest systemic NOEL found in a chronic or subchronic study of dogs, rats, or mice. Reproductive effects, including the risk of birth defects, are evaluated based on the lowest maternal toxic, fetotoxic, or teratogenic NOELs found in a two- or three-generation reproduction study or in a teratology study.

For members of the public, MOSs were computed for each herbicide used in each program for routine-realistic and routine-worst case situations, including single and multiple routes of exposure. MOSs for workers were computed for each herbicide, program, and worker category for routine-realistic and routine-worst case exposures. Accident scenarios were also evaluated. The MOSs were computed by comparing the laboratory-determined NOELs in Table E5-1 with the estimated doses.

Cancer Risk

Assumptions Used in the Cancer Risk Analysis

As a result of the review of cancer studies presented in Section E3, a risk analysis for cancer was conducted for eight of the herbicides (amitrole, atrazine, bromacil, 2,4-D, glyphosate, picloram, and simazine) and the carriers diesel oil and kerosene, because they may be oncogenic.

Cancer risks for these 10 chemicals were calculated based on the following conservative assumptions that are likely to exaggerate the risks:

(1) Even though some have given equivocal laboratory results as to oncogenicity, a cancer risk analysis was conducted on all 10, to provide a worst case assessment of risk.

(2) In cases where there is more than one tumor data set available, the data set indicating greater carcinogenic potency was chosen to compute risk.

(3) It is assumed that carcinogenicity is not a threshold phenomenon; that is, any dose of these chemicals has some probability of causing cancer, no matter how small the dose.

(4) Interspecies extrapolation from test animals to humans is a major source of uncertainty in judging cancer risk. The scaling method used in this analysis is the most conservative of the commonly accepted methods. The cancer potency of each chemical for humans was assumed to be the same as the potency for the laboratory animals when scaled in terms of body surface area. This method is recommended by EPA and others (NRC 1986), but it is not the only acceptable approach. Another acceptable method (OSTP 1985) is to scale doses directly to body weight,

resulting in estimates of cancer risk that are about 16 percent of those calculated here.

(5) The range of doses calculated for the public and workers in the routine scenarios covers even worst case exposures that might be encountered with each application method. Unusual exposure situations, represented by accidental spraying and large herbicide spills, have also been considered.

Cancer Risk Probability Calculations

The probability of cancer occurrence over a lifetime as a result of exposure to each of the chemicals was calculated using the following equations:

$$P(d) = 1 - e^{(-K \times b \times d)}$$

$$d = D \times N/L$$

where:

$P(d)$ = a conservative estimate of the probability of cancer during a person's lifetime as the result of dose d

d = the average daily dose over a lifetime (mg/kg/day)

b = a conservative estimate for cancer potency in the test animal

K = an interspecies extrapolation factor, used to correct b for application to humans

D = the daily dose (mg/kg/day)

N = the number of days during which the dose D occurs during an individual's lifetime

L = the number of days in a lifetime, taken to be 25,550 for a 70-year life span

The cancer potency values have units of $(mg/kg/day)^{-1}$, as they are a probability that a tumor will be produced as a result of daily exposure to one milligram per kilogram body weight of the chemical, that is, the likelihood of cancer per unit daily dose.

In this risk assessment, and in Table E5-1, the cancer potency values reported are already adjusted for extrapolation from laboratory

Table E5-1. Toxicity Reference Levels Used in the Risk Analysis

Herbicide	Systemic NOEL (mg/kg/day)	Reproductive NOEL (mg/kg/day)	Cancer Potency (mg/kg/day)[-1]
Amitrole	0.025	4.00	1.4
Atrazine	0.38	0.5	0.22
Bromacil	6.25	12.5	0.0038
Chlorsulfuron	5.0	25.0	N[a]
Clopyralid	15	75	N
2,4-D	1.0	5.0	0.029
Dalapon	8.0	12.5	N
Dicamba	15.8	3.0	N
Diuron	0.625	6.25	N
Glyphosate	31	10	0.000024
Hexazinone	10	50	N
Imazapyr	500	300	N
Mefluidide	1.5	60	N
Metsulfuron methyl	1.25	25	N
Picloram	7.0	50	0.003
Simazine	5.0	5.0	0.083
Sulfometuron methyl	2.5	25	N
Tebuthiuron	12.5	5.0	N
Triclopyr	2.5	10	N
Carriers			
Diesel oil	7.38	751	0.0000049
Kerosene	28	751	0.0000049

[a]Not a possible or proven carcinogen.

animals to humans, and thus represent b x K from the preceding equation.

The interspecies extrapolation factor, K, can be estimated by assuming that body surface area is proportional to body weight to the 2/3 power (Mantel and Schneiderman 1975). Extrapolation to humans from animal doses expressed as mg/kg uses the following equation:

$$K = (\text{human weight/test animal weight})^{1/3}$$

For an average human weight of 70 kg and an average rat weight of 350 g, K is estimated to be 5.8. For mice weighing 25 g, K is 14.1. These values were used in the analysis, although lesser values would apply if a smaller human weight were assumed.

Public Lifetime Dose Estimation

Cancer risk from the herbicides and carriers for members of the public was calculated assuming four exposures per year over 5 years as the total lifetime exposure due to BLM's use of the chemicals. Individual exposure routes were considered separately in estimating risk from cumulative exposures. It was assumed that the realistic dose accounted for 95 percent of the total dose, and the worst case dose accounted for the remaining 5 percent.

Worker Lifetime Dose Estimation

Cancer risk to workers from the herbicides and carriers was calculated assuming that a worker is employed in herbicide application for 10 years and receives the realistic dose 90 percent of the working time and the worst case dose the remaining 10 percent.

Comparison of Cancer Risks With Other Common Risks

To put the estimated cancer risks in perspective, risks associated with some more familiar hazards and occupational risks are listed in Table E5-2. A variety of hazards are listed in the table that have a risk of about 1 in 1 million. They include smoking 2 cigarettes, eating 6 pounds of peanut butter, drinking 40 sodas sweetened with saccharin, or taking 1 transcontinental round trip by air.

Table E5-2. Risk of Death Resulting From Common Activities and Occupations for Persons Living in the United States

Activity	Time to Accumulate a 1-In-1-Million Risk of Death	Lifetime Risk per Capita[a]
Accidents or Catastrophes		
Motor Vehicle Accident	1.5 days	1×10^{-2}
Falls	6 days	4×10^{-3}
Drowning	10 days	3×10^{-3}
Fires	13 days	2×10^{-3}
Firearms	36 days	7×10^{-4}
Electrocution	2 months	4×10^{-4}
Tornados	20 months	4×10^{-5}
Floods	20 months	4×10^{-5}
Lightning	2 years	3×10^{-5}
Animal Bite or Sting	4 years	2×10^{-5}
Everyday Risks		
Eating and Drinking[b]	6 pounds of peanut butter (aflatoxin)	
40 Diet Sodas (Saccharin)		
90 Pounds of Broiled Steak (Cancer Risk Only)		
180 Pints of Milk (Aflatoxin)		
200 Gallons of Drinking Water From Miami or New Orleans		
Smoking	2 cigarettes	
Occupational Risks		
General		
Mining and Quarrying	9 hours	3×10^{-2}
Construction	14 hours	2×10^{-2}
Agriculture	15 hours	2×10^{-2}
Transport/Public Util.	1 day	1×10^{-2}
Service/Government	3.5 days	3×10^{-3}
Manufacturing	4.5 days	2×10^{-3}
Trade	7 days	1×10^{-3}
Specific		
Firefighting	11 hours	2×10^{-2}
Coal Mining	14 hours	2×10^{-2}
Police Duty	1.5 days	6×10^{-3}
Railroad Employment	1.5 days	6×10^{-3}

[a]Assuming 30 years at risk for occupational risks, 70 years at risk for other risks.
[b]Amount needed to accumulate a 1-in-1 million risk of death.
Source: Adapted from Crouch and Wilson (1982).

The cancer risk for a single x ray is 7 in 1 million. Many occupational risks are greater. Working for 30 years in agriculture or construction has a risk of about 2 in 100; and mining and quarrying have an even greater risk of 3 in 100 over 30 years.

Health Risks for the BLM Programs

This section presents the results of the risk analysis for the herbicides and carriers proposed for use on BLM-managed lands in the 13 Western States. The estimated exposures on which the risk estimates are based were calculated using the herbicide application information and methods described in Section E4. The margins of safety and cancer risk values are based on the methods described previously in this section. The risks that exceed the risk criteria (MOS less than 100 or cancer risk greater than 1 in 1 million) are summarized in Tables E5-3 through E5-17 (at the end of this section) for each program for members of the public and workers. In the following sections, risks are discussed only for those scenarios in which the risks exceed these criteria. The numbers in parentheses that accompany the discussion are MOSs that are less than 100 for systemic and reproductive effects or cancer risk estimates that exceed 1 in 1 million.

Risks From Rangeland Herbicide Treatments

Those applications that present a significant risk from herbicide use on rangeland under the BLM program are summarized in Table E5-3 for members of the public, E5-4 for workers, and E5-5 for accidents. The herbicides used on rangeland are amitrole, atrazine, clopyralid, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, imazapyr, picloram, tebuthiuron, and triclopyr, as well as the carriers diesel oil and kerosene.

Risks to Members of the Public

Aerial Applications. Routine-realistic aerial applications of the BLM herbicides present few risks to members of the public. The MOS is less than 100 for systemic effects from eating fish from a body of water that has received amitrole spray drift (38) and for the cumulative exposure that an angler may receive from amitrole exposure (32). Routine-worst case aerial applications present a risk of systemic effects from drinking water that has received amitrole spray drift (33); from eating fish from

a body of water that has been contaminated with drift from nearby amitrole (7) or 2,4-D (89) applications; from cumulative exposure to amitrole by a hiker (33), berrypicker (25), or angler (5); and from cumulative exposure to 2,4-D by an angler (64).

No routine aerial applications of the herbicides on rangeland present a significant risk of adverse reproductive or teratogenic effects to members of the public.

An angler's cumulative exposure to amitrole results in a risk of cancer that slightly exceeds the cancer probability risk criterion of 1 in 1 million (1.08 in 1 million).

Backpack Applications. Realistic backpack applications of herbicides on rangeland do not present any significant risks to members of the public.

There are no significant risks of reproductive or teratogenic effects to members of the public from backpack applications of the BLM herbicides on rangeland.

No cancer risk estimate exceeds 1 in 1 million for a member of the public in this scenario.

Ground Mechanical Applications. Routine-realistic and routine-worst case ground mechanical applications of amitrole present a risk of systemic effects from vegetation contact by a berrypicker (28 and 8, respectively) and from the cumulative exposure of a berrypicker (28 and 8).

No significant adverse reproductive effects were predicted for members of the public from ground mechanical applications on rangeland.

Vegetation contact by a berrypicker may result in a significant cancer risk (1.09 in 1 million), as may the cumulative exposure received by a berrypicker (1.11 in 1 million).

Hand Applications. BLM does not use these methods on rangeland.

Risks to Workers

Aerial Applications. Imazapyr and picloram risk estimates for workers in aerial applications result in MOSs greater than 100 in both the routine-realistic case and routine-worst case for all aerial application worker categories. Imazapyr is not considered carcinogenic in this risk assessment. Although picloram may be a potential carcinogen, cancer risk estimates are less than 1 in 1 million for all workers in aerial rangeland herbicide applications.

Routine-realistic aerial applications of herbicides to BLM-managed rangeland may result in significant risks of systemic effects to pilots from amitrole (74), atrazine (23), 2,4-D (16), and triclopyr (99) and to mixer-loaders from amitrole (30), atrazine (9), 2,4-D (7), dalapon (63), dicamba (94), tebuthiuron (74), triclopyr (40), and diesel oil (88). No high systemic risks for fuel truck operators are expected as a result of routine-realistic aerial applications. In the routine-worst case, there are significant risks to pilots from amitrole (5), atrazine (1), 2,4-D (1), dalapon (10), dicamba (15), glyphosate (23), hexazinone (56), tebuthiuron (12), triclopyr (6), diesel oil (14), and kerosene (52); to mixer-loaders from amitrole (4), atrazine (1), clopyralid (88), 2,4-D (-1), dalapon (8), dicamba (12), glyphosate (18), hexazinone (44), tebuthiuron (9), triclopyr (5), diesel oil (11), and kerosene (41); and to fuel truck operators from atrazine (46) and 2,4-D (34).

In the routine-realistic case, significant reproductive risks are present for pilots from the use of atrazine (30), 2,4-D (82), dicamba (44), and tebuthiuron (74) and for mixer-loaders from atrazine (12), 2,4-D (33), dalapon (99), dicamba (18), glyphosate (47), and tebuthiuron (30). There are no high reproductive risks to fuel truck operators under realistic conditions. In the routine-worst case, there are significant adverse reproductive risks to pilots from atrazine (2), 2,4-D (5), dalapon (16), dicamba (3), glyphosate (7), tebuthiuron (5), and triclopyr (25); to mixer-loaders from atrazine (1), 2,4-D (4), dalapon (12), dicamba (2), glyphosate (6), tebuthiuron (4), and triclopyr (20); and to fuel truck operators from atrazine (60) and dicamba (91).

Cancer risks exceed 1 in 1 million for pilots from amitrole (2.76 in 1 million), atrazine (2.17 in 100 thousand), and 2,4-D (1.03 in 100 thousand) and for mixer-loaders from amitrole (4.74 in 1 million), atrazine (3.72 in 100 thousand), and 2,4-D (1.77 in 100 thousand). No estimated cancer risks for fuel truck operators in rangeland aerial herbicide applications exceed 1 in 1 million.

Backpack Applications. Backpack applicators are not expected to face any significant systemic, reproductive, or cancer risks from the use of clopyralid, hexazinone, imazapyr, picloram, tebuthiuron, or kerosene on rangeland.

Routine-realistic backpack applications of herbicides to BLM-managed rangeland are not expected to result in significant systemic risks to applicators. However, in the routine-worst

case scenario, there are high systemic risks from amitrole (23), atrazine (7), 2,4-D (8), dalapon (48), triclopyr (30), and diesel oil (67).

There are no significant reproductive risks to backpack applicators applying herbicides to rangeland in the realistic case. In the worst case, there are notable risks from atrazine (9), 2,4-D (38), dalapon (76), dicamba (27), and glyphosate (45).

Cancer risk estimates are significant for applicators using atrazine (3.77 in 1 million) or 2,4-D (1.19 in 1 million) on rangeland.

Ground Mechanical Applications. No excess systemic, reproductive, or cancer risks to workers from rangeland herbicide application by ground mechanical methods are expected to result from the use of clopyralid, hexazinone, imazapyr, picloram, or kerosene.

For workers using ground mechanical equipment to apply herbicides to rangeland, there are significant systemic risks in the routine-realistic case for applicators from atrazine (71) and 2,4-D (78); for mixer-loaders from amitrole (98), atrazine (30), and 2,4-D (33), and for applicator/mixer-loaders from atrazine (31) and 2,4-D (34). In the worst case, there are high risks to applicators from amitrole (9), atrazine (3), 2,4-D (2), dalapon (20), dicamba (30), glyphosate (47), tebuthiuron (24), triclopyr (13), and diesel oil (28); to mixer-loaders from amitrole (23), atrazine (7), 2,4-D (5), dalapon (48), dicamba (72), tebuthiuron (57), triclopyr (30), and diesel oil (67); and to applicator/mixer-loaders from amitrole (17), atrazine (5), 2,4-D (4), dalapon (36), dicamba (53), glyphosate (83), tebuthiuron (42), triclopyr (22), and diesel oil (50).

In the realistic case, there are significant reproductive risks from atrazine to applicators (93), mixer-loaders (39), and applicator/mixer-loaders (40). In the worst case, high reproductive risks are expected for applicators from atrazine (4), 2,4-D (10), dalapon (31), dicamba (6), glyphosate (15), tebuthiuron (9), and triclopyr (50); for mixer-loaders from atrazine (9), 2,4-D (25), dalapon (76), dicamba (14), glyphosate (36), and tebuthiuron (23); and for applicator/mixer-loaders from atrazine (7), 2,4-D (19), dalapon (56), dicamba (10), glyphosate (27), tebuthiuron (17), and triclopyr (90).

There are significant cancer risks from ground mechanical rangeland herbicide application for applicators from atrazine (7 in 1 million) and 2,4-D (3.03 in 1 million), for mixer-loaders from

atrazine (6.57 in 1 million) and 2,4-D (2.42 in 1 million), and for applicator/mixer-loaders from atrazine (7.21 in 1 million) and 2,4-D (2.74 in 1 million).

Hand Applications. Hand application of herbicides is not used on BLM-managed rangeland.

Risks From Accidents

Systemic margins of safety are below 100 for most herbicides used on rangeland for accidents. The highest systemic risks are from an accidental spill of the herbicide concentrate on the skin, where all MOSs from the herbicides used on rangeland are less than 100 and all doses except those calculated for imazapyr and picloram exceed the NOEL, resulting in a negative MOS. A spill on the skin of herbicide mixture results in systemic risks almost as high as a spill of the concentrate. Direct spray of a person results in systemic MOSs less than 100 for amitrole, atrazine, 2,4-D, dalapon, dicamba, tebuthiuron, triclopyr, and diesel oil. Ingestion of water that has been contaminated by a direct spray results in systemic MOSs less than 100 for amitrole and 2,4-D. Eating fish from this same body of water results in significant risks of systemic effects for amitrole, 2,4-D, and triclopyr. Immediate reentry to a sprayed area by a hiker does not present a significant risk of systemic effects. However, immediate reentry by a berrypicker poses significant systemic risks from amitrole, atrazine, 2,4-D, dalapon, dicamba, glyphosate, tebuthiuron, triclopyr, and diesel oil. Eating directly sprayed berries results in systemic MOSs less than 100 from amitrole, atrazine, and 2,4-D. Drinking water that has been contaminated by a jettison of herbicide mixture from a helicopter presents significant systemic risks from all chemicals used on rangeland except for clopyralid, glyphosate, hexazinone, imazapyr, and kerosene. Drinking water that has been contaminated by a batch truck spill of herbicide mixture results in systemic MOSs less than 100 for all herbicides except imazapyr.

Rangeland accident scenarios also may result in several significant risks to reproductive health and fetal development. A spill of concentrate or mixture on the skin presents significant risks from all herbicides used on rangeland. Direct spray of a person may lead to significant reproductive risks from atrazine, 2,4-D, dicamba, glyphosate, and tebuthiuron. Drinking directly sprayed water is not expected to pose significant reproductive risks. However, eating fish from that same body of

water may result in significant risks from 2,4-D and dicamba. Although immediate reentry by a hiker is not likely to result in any significant risks, reproductive MOSs for immediate reentry by a berrypicker are less than 100 for atrazine, 2,4-D, dalapon, dicamba, glyphosate, tebuthiuron, and triclopyr. Eating directly sprayed berries entails significant reproductive risk from atrazine, 2,4-D, dicamba, and tebuthiuron. Drinking water contaminated by a helicopter jettison of herbicide mixture has a significant reproductive risk from amitrole, atrazine, 2,4-D, dalapon, dicamba, glyphosate, and tebuthiuron. Drinking water that has been contaminated by a batch truck spill poses significant reproductive risks from all chemicals except clopyralid, imazapyr, diesel oil, and kerosene.

Cancer risks that exceed 1 in 1 million are predicted for amitrole, atrazine, and 2,4-D from a spill of either concentrate or mixture on the skin; for amitrole from eating fish that came from directly sprayed water; for amitrole from eating berries that were directly sprayed; for amitrole from drinking water contaminated by a helicopter jettison of mixture; and for amitrole, atrazine, and 2,4-D from drinking water that was contaminated by a batch truck spill.

Risks From Public-Domain Forest Land Herbicide Treatments

Scenarios in which the MOSs are less than 100 or cancer risk probabilities are greater than 1 in 1 million are summarized in Table E5-6 for members of the public, Table E5-7 for workers, and Table E5-8 for accidents. The herbicides used on public-domain forest lands are amitrole, atrazine, chlorsulfuron, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, imazapyr, picloram, simazine, tebuthiuron, and triclopyr, as well as the carriers diesel oil and kerosene.

Risks to Members of the Public

Aerial Applications. Routine realistic aerial application of BLM herbicides to public-domain forest land may present a significant risk of adverse systemic effects to members of the public from eating fish from a body of water that has received amitrole spray drift (38) and from the multiple exposures to amitrole that an angler may receive (32). Worst case aerial applications pose elevated systemic risks to berrypickers from vegetation contact with foliage contaminated by atrazine spray drift (97); drinking water contaminated by amitrole spray drift (33); eating fish from a body of water contaminated by spray drift from amitrole (7) or 2,4-D (67); the multiple exposures to

amitrole that a hiker may receive (33); the multiple exposures that a berrypicker may have to amitrole (25), atrazine (51), or 2,4-D (96); and the multiple exposures that an angler may have to amitrole (5), atrazine (76), or 2,4-D (48).

Members of the public are not expected to have any significant reproductive risks from routine realistic aerial application of the BLM herbicides to public-domain forest land. However, in the routine-worst case, there is a significant risk to berrypickers who may be exposed through several routes to atrazine (68).

Single routes of exposure are unlikely to result in a significant cancer risk to members of the public from aerial applications. The multiple exposures received by an angler may lead to a significant cancer risk from amitrole (1.08 in 1 million).

Backpack Applications. Estimated systemic MOSs for members of the public for routine-realistic exposures in this scenario are all greater than 100.

There are no significant reproductive risks to members of the public from routine-realistic exposures in this scenario.

There are no significant cancer risks to members of the public from backpack applications of herbicides on BLM-managed public-domain forest land.

Ground Mechanical Applications. In the routine-realistic case, members of the public may have a risk of adverse systemic effects from the use of ground mechanical herbicide application from amitrole from the vegetation contact that a berrypicker may have (28) and the multiple exposures that a berrypicker may receive (28). In the routine-worst case, there is a significant risk of systemic effects from vegetation contact by a berrypicker from amitrole (8), atrazine (61), and 2,4-D (81), and the multiple exposures that a berrypicker may have to amitrole (8), atrazine (58), and 2,4-D (77).

In the routine-realistic case, there are no significant reproductive risks from the ground mechanical herbicide application to members of the public. In the routine-worst case, there is a significant risk of reproductive effects from atrazine from the vegetation contact that a berrypicker may have (81) and the multiple exposures of a berrypicker (76).

A significant risk of cancer exists from amitrole from the vegetation contact that a berrypicker may have (1.09 in 1 million) and the multiple exposures of a berrypicker (1.11 in 1 million).

Hand Applications. No significant risks of systemic effects, reproductive effects, or cancer are expected for members of the public as a result of hand applications of herbicides to BLM-managed public-domain forest land.

Risks to Workers

Aerial Applications. MOSs are greater than 100 and cancer risks less than 1 in 1 million for workers aerially applying chlorsulfuron, imazapyr, picloram, and kerosene to BLM-managed public-domain forest land.

In the routine-realistic case, there are significant risks to workers of adverse systemic effects to pilots from atrazine (36) or 2,4-D (79) and to mixer-loaders from atrazine (14), 2,4-D (32), or triclopyr (95). MOSs are all above 100 for fuel truck operators in the realistic case. In the routine-worst case, there are significant systemic risks to pilots from amitrole (15), atrazine (1), 2,4-D (2), dalapon (24), dicamba (94), hexazinone (40), simazine (15), tebuthluron (30), triclopyr (7), and diesel oil (44); to mixer-loaders from amitrole (12), atrazine (-1), 2,4-D (2), dalapon (19), dicamba (74), glyphosate (97), hexazinone (31), simazine (12), tebuthluron (23), triclopyr (6), and diesel oil (35); and to fuel truck operators from atrazine (37) and 2,4-D (81).

In the routine-realistic case, aerial herbicide application to public-domain forest land may result in significant reproductive risks from atrazine to pilots (47) and mixer-loaders (19). Fuel truck operators' MOSs are all above 100 under realistic conditions. In the routine-worst case, there are significant reproductive risks to pilots from atrazine (1), 2,4-D (12), dalapon (37), dicamba (18), glyphosate (40), simazine (15), tebuthluron (12), and triclopyr (30); to mixer-loaders from atrazine (1), 2,4-D (10), dalapon (29), dicamba (14), glyphosate (31), simazine (12), tebuthluron (9), and triclopyr (23); and to fuel truck operators from atrazine (48).

In this scenario, cancer risks exceed 1 in 1 million for pilots for atrazine (2.22 in 100 thousand), 2,4-D (3.51 in 1 million), and simazine (8.38 in 1 million) and for mixer-loaders for amitrole (1.09 in 1 million), atrazine (3.43 in 100 thousand), 2,4-D (5.42 in 1 million), and simazine (1.29 in 100 thousand). Cancer risks for fuel truck operators are greater than 1 in 1 million.

Backpack Applications. No significant systemic, reproductive, or cancer risks are predicted for backpack applicators applying herbicides in BLM-managed public-domain forest land from chlorsulfuron, imazapyr, picloram, or kerosene.

In the routine-realistic case, backpack applicators have a notable systemic risk from atrazine (26). In the routine-worst case, there are significant systemic risks from amitrole (23), atrazine (2), 2,4-D (8), dalapon (36), hexazinone (91), simazine (23), triclopyr (23), and diesel oil (67).

Reproductive risk is present for applicators in the realistic case from atrazine (35). In the worst case, high reproductive risks are posed by atrazine (2), 2,4-D (38), dalapon (57), dicamba (27), glyphosate (91), simazine (23), tebuthiuron (61), and triclopyr (91).

Significant cancer risks are present for applicators from atrazine (1.51 in 100 thousand), 2,4-D (1.19 in 1 million), and simazine (5.69 in 1 million).

Ground Mechanical Applications. Workers using ground mechanical equipment to treat BLM-managed public-domain forest lands are not expected to have any significant systemic, reproductive, or cancer risks from the use of chlorsulfuron, imazapyr, picloram, or kerosene.

The use of ground mechanical equipment to apply herbicides on public-domain forest land results in systemic risks to applicators from atrazine (43), to mixer-loaders from atrazine (18) and 2,4-D (79), and to applicator/mixer-loaders from atrazine (18), and 2,4-D (81) in the routine-realistic case. Using worst case assumptions, significant systemic risks are posed for applicators from amitrole (9), atrazine (-1), 2,4-D (2), dalapon (15), dicamba (60), glyphosate (78), hexazinone (25), simazine (9), tebuthiuron (19), triclopyr (5), and diesel oil (28); for mixer-loaders from amitrole (23), atrazine (2), 2,4-D (4), dalapon (36), hexazinone (60), simazine (23), tebuthiuron (45), triclopyr (11), and diesel oil (67); and to applicator/mixer-loaders from amitrole (17), atrazine (1), 2,4-D (3), dalapon (27), hexazinone (45), simazine (17), tebuthiuron (34), triclopyr (8), and diesel oil (50).

In the routine-realistic case, atrazine poses significant reproductive risks for applicators (56), mixer-loaders (24), and applicator/mixer-loaders (24). In the worst case, there are significant reproductive risks for applicators from atrazine (-1), 2,4-D (8), dalapon (24),

dicamba (11), glyphosate (25), simazine (9), tebuthiuron (8), and triclopyr (19); to mixer-loaders from atrazine (2), 2,4-D (19), dalapon (57), dicamba (27), glyphosate (60), simazine (23), tebuthiuron (18), and triclopyr (45); and to applicator/mixer-loaders from atrazine (2), 2,4-D (14), dalapon (42), dicamba (20), glyphosate (45), simazine (17), tebuthiuron (13), and triclopyr (34).

For ground mechanical treatment of public-domain forest lands, worker cancer risks exceed 1 in 1 million for applicators from atrazine (2.37 in 100 thousand), 2,4-D (3.5 in 1 million), and simazine (8.93 in 1 million); for mixer-loaders from atrazine (1.59 in 100 thousand), 2,4-D (1.94 in 1 million), and simazine (6.01 in 1 million); and for applicator/mixer-loaders from atrazine (1.87 in 100 thousand), 2,4-D (2.93 in 1 million), and simazine (7.07 in 1 million).

Hand Applications. The hand applicator on BLM-managed public-domain forest land is not expected to face any significant systemic, reproductive, or cancer risks from the use of hexazinone, imazapyr, picloram, or kerosene.

In the routine-realistic case, workers using hand equipment to treat public-domain forest land with herbicides may have notable systemic risks from the use of atrazine (15), 2,4-D (65), or triclopyr (97). In the routine-worst case, systemic risks are high to hand applicators from amitrole (35), atrazine (3), chlorsulfuron (35), 2,4-D (12), dalapon (19), simazine (35), tebuthiuron (29), triclopyr (17), and diesel oil (52).

Routine-realistic reproductive MOSs are less than 100 for hand applicators using atrazine (19) or tebuthiuron (65). In the worst case, there are high reproductive risks from atrazine (3), 2,4-D (58), dalapon (30), dicamba (42), glyphosate (93), simazine (35), tebuthiuron (12), and triclopyr (70).

Cancer risks exceed 1 in 1 million for the hand applicator on public-domain forest land from atrazine (2.26 in 100 thousand), 2,4-D (1.79 in 1 million), and simazine (8.52 in 1 million).

Accidents

Significant systemic risks are likely from a skin spill of herbicide concentrate or mixture of any of the herbicides used on public-domain forest land. In most cases, the estimated dose exceeds the laboratory-determined NOEL. Direct spray of a person results in significant systemic risks from amitrole, atrazine, 2,4-D, dalapon, hexazinone, simazine, tebuthiuron,

triclopyr, and diesel oil. Drinking water that has been directly sprayed may cause high systemic risks from amitrole, atrazine, and 2,4-D. Eating fish from a body of water that has been directly sprayed is expected to pose a risk from amitrole, atrazine, 2,4-D, simazine, and triclopyr. There are no higher risks from immediate reentry to a treated area by a hiker. However, immediate reentry by a berrypicker may lead to elevated systemic risks from amitrole, atrazine, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, simazine, tebuthiuron, triclopyr, and diesel oil. Eating berries that have been directly sprayed may result in significant systemic risks from amitrole, atrazine, 2,4-D, simazine, or triclopyr. Drinking water that has been contaminated by a helicopter jettison of herbicide mixture results in high risks of systemic effects from all herbicides and carriers except for chlorsulfuron, glyphosate, imazapyr, and kerosene. Drinking water that has been contaminated by a batch truck spill of herbicide mixture results in a significant systemic risk from all chemicals except imazapyr.

Reproductive risks are significant for all herbicides from a skin spill of herbicide concentrate or mixture. Direct spray of a person leads to a high reproductive risk from atrazine, 2,4-D, dalapon, dicamba, glyphosate, simazine, tebuthiuron, and triclopyr. Drinking directly sprayed water presents a reproductive risk from atrazine. Eating fish from a directly sprayed body of water leads to a high reproductive risk from atrazine, 2,4-D, dicamba, and simazine. Immediate reentry by a hiker is not expected to result in any adverse reproductive effects. Immediate reentry by a berrypicker is expected to have a risk to reproductive indices from atrazine, 2,4-D, dalapon, dicamba, glyphosate, simazine, tebuthiuron, and triclopyr. Eating berries that have been directly sprayed is likely to cause a high reproductive risk from atrazine, 2,4-D, dicamba, simazine, and tebuthiuron. Drinking water that has been contaminated by a helicopter jettison of herbicide mixture may result in significant reproductive risks from amitrole, atrazine, 2,4-D, dalapon, dicamba, glyphosate, simazine, tebuthiuron, and triclopyr. Drinking water that has been contaminated by a batch truck spill of herbicide mixture results in significant reproductive risks from amitrole, atrazine, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, picloram, simazine, tebuthiuron, and triclopyr.

Risks of cancer that exceed 1 in 1 million were estimated for amitrole, atrazine, 2,4-D, and

simazine from a skin spill of herbicide concentrate or mixture and drinking water that has been contaminated by a batch truck spill. Direct spray of a person results in a significant cancer risk from atrazine. Drinking directly sprayed water is not expected to result in a high cancer risk. However, eating fish from a directly sprayed body of water poses an elevated risk of cancer from amitrole. Immediate reentry by a hiker does not result in excess cancer risk, but immediate reentry by a berrypicker does, from atrazine and simazine. Eating directly sprayed berries presents a high cancer risk from amitrole. Drinking water that has been contaminated by a helicopter jettison of herbicide mixture is estimated to cause a high cancer risk from amitrole and atrazine. Drinking water that has been contaminated by a batch truck spill presents a high risk from amitrole, atrazine, 2,4-D, and simazine.

Oil and Gas Sites

Significant risks from herbicide applications on BLM-managed oil and gas sites are presented in Table E5-9 for members of the public, E5-10 for workers, and E5-11 for accidents. The herbicides used on oil and gas sites are amitrole, atrazine, bromacil, chlorsulfuron, clopyralid, 2,4-D, dalapon, dicamba, diuron, glyphosate, hexazinone, imazapyr, mefluidide, metsulfuron methyl, picloram, simazine, sulfometuron methyl, tebuthiuron, and triclopyr, and the carriers diesel oil and kerosene.

Risks to Members of the Public

Aerial Applications. Routine-realistic aerial applications of herbicides on oil and gas sites are not expected to result in any significant risks of systemic effects to members of the public. Routine-worst case applications may result in significant risks from dermal exposure to spray drift of atrazine (25) and diuron (52); the multiple exposures of a hiker to atrazine (25) and diuron (51), and the multiple exposures of a nearby resident to atrazine (25) and diuron (51).

Routine-realistic aerial application to oil and gas sites is not expected to result in any significant reproductive risks to members of the public. However, in the routine-worst case, there are significant reproductive risks from dermal exposure to spray drift from atrazine (33) and from the multiple exposures to atrazine that may be received by a hiker (33) or a nearby resident (33).

Estimated cancer risk probabilities for members of the public as a result of aerial applications

of herbicides on BLM-managed oil and gas sites do not exceed 1 in 1 million.

Backpack Applications. Routine-realistic backpack applications of herbicides on BLM-managed oil and gas sites are not expected to result in any adverse systemic effects for members of the public.

No significant reproductive effects for members of the public are expected from routine-realistic backpack applications on oil and gas sites.

Cancer risks estimated for members of the public as a result of oil and gas site backpack herbicide application do not exceed 1 in 1 million.

Ground Mechanical Applications. There are not expected to be any significant systemic or reproductive risks to members of the public from ground mechanical herbicide application on BLM-managed oil and gas sites. No cancer risks in this scenario exceed 1 in 1 million.

Hand Applications. No significant systemic, reproductive, or cancer risks to members of the public are expected from hand application of herbicides to oil and gas sites.

Risks to Workers

Aerial Applications. Herbicides used in aerial oil and gas site applications for which no worker is estimated to have an MOS less than 100 or a cancer risk greater than 1 in 1 million are chlorsulfuron, imazapyr, mefluidide, metsulfuron methyl, picloram, and kerosene.

Routine-realistic aerial application of herbicides to oil and gas sites may cause significant systemic risks to pilots from amitrole (96), atrazine (4), diuron (7), and simazine (47) and to mixer-loaders from amitrole (38), atrazine (1), bromacil (59), 2,4-D (63), dalapon (55), diuron (3), simazine (19), and triclopyr (47). There are no significant adverse systemic risks to fuel truck operators in the realistic case. In the routine-worst case, there are significant systemic risks to pilots from amitrole (6), atrazine (-4), bromacil (9), clopyralid (30), 2,4-D (10), dalapon (9), dicamba (94), diuron (-2), hexazinone (22), simazine (3), tebuthiuron (19), triclopyr (7), and diesel oil (88); to mixer-loaders from amitrole (5), atrazine (-6), bromacil (7), clopyralid (23), 2,4-D (8), dalapon (7), dicamba (14), diuron (-3), hexazinone (17), simazine (2), sulfometuron methyl (83), tebuthiuron (15), triclopyr (6), and diesel oil (69); and to fuel truck operators from atrazine (7), diuron (15), and simazine (97).

Under the routine-realistic case, significant reproductive risks exist for pilots from atrazine (5), diuron (74), and simazine (47) and for mixer-loaders from atrazine (2), dalapon (86), diuron (30), simazine (19), and tebuthiuron (47). There are no high reproductive risks for fuel truck operators in the realistic case. In the routine-worst case, there are significant risks to pilots from atrazine (-3), bromacil (19), 2,4-D (50), dalapon (14), dicamba (18), diuron (5), glyphosate (60), simazine (3), tebuthiuron (7), and triclopyr (30); to mixer-loaders from atrazine (-4), bromacil (15), 2,4-D (39), dalapon (11), dicamba (14), diuron (4), glyphosate (47), hexazinone (87), simazine (2), tebuthiuron (6), and triclopyr (23); and to fuel truck operators from atrazine (10), and simazine (97).

For workers involved in aerial herbicide applications on oil and gas sites, cancer risks are significant for pilots from amitrole (2.13 in 1 million), atrazine (1.36 in 10 thousand), 2,4-D (1.07 in 1 million), and simazine (5.11 in 100 thousand); for mixer-loaders from amitrole (3.66 in 1 million), atrazine (2.33 in 10 thousand), bromacil (1.61 in 1 million), 2,4-D (1.84 in 1 million), and simazine (8.78 in 100 thousand); and for fuel truck operators from atrazine (4.25 in 1 million) and simazine (1.60 in 1 million).

Backpack Applications. No significant systemic, reproductive, or cancer risks are expected for backpack applicators on oil and gas sites applying chlorsulfuron, imazapyr, metsulfuron methyl, picloram, or kerosene.

In the routine-realistic case, backpack applicators on oil and gas sites have significant systemic risks from atrazine (11) and diuron (17). In the worst case, they have high systemic risks from amitrole (9), atrazine (-2), bromacil (11), clopyralid (18), 2,4-D (6), dalapon (29), dicamba (57), diuron (-1), hexazinone (36), mefluidide (87), simazine (7), sulfometuron methyl (65), tebuthiuron (23), triclopyr (9), and diesel oil (54).

Backpack applicators have high reproductive risks from atrazine (14) in the realistic case. In the worst case, reproductive risks are significant from atrazine (-1), bromacil (23), clopyralid (91), 2,4-D (30), dalapon (45), dicamba (11), diuron (9), glyphosate (36), simazine (7), tebuthiuron (9), and triclopyr (36).

Cancer risks to backpack applicators on oil and gas sites exceed 1 in 1 million for amitrole (1.11 in 1 million), atrazine (4.36 in 100

thousand), 2,4-D (1.38 in 1 million), and simazine (1.65 in 100 thousand).

Ground Mechanical Applications. No significant systemic, reproductive, or cancer risks are expected for workers using ground mechanical equipment on oil and gas sites to apply chlorsulfuron, imazapyr, metsulfuron methyl, picloram, or kerosene.

Routine-realistic exposures to workers in ground mechanical oil and gas site applications presents significant risks of systemic effects to applicators from atrazine (21) and diuron (35); to mixer-loaders from atrazine (9), 2,4-D (98), and diuron (15); and to applicator/mixer-loaders from atrazine (9) and diuron (15). Worst case exposures result in high systemic risks to applicators from amitrole (4), atrazine (-7), bromacil (6), clopyralid (19), 2,4-D (6), dalapon (5), dicamba (60), diuron (-3), hexazinone (14), mefluidide (90), simazine (2), sulfometuron methyl (67), tebuthiuron (12), triclopyr (5), and diesel oil (56); to mixer-loaders from amitrole (9), atrazine (3), bromacil (14), clopyralid (45), 2,4-D (15), dalapon (13), diuron (-1), hexazinone (34), simazine (5), tebuthiuron (28), and triclopyr (11); and to applicator/mixer-loaders from amitrole (7), atrazine (-4), bromacil (10), clopyralid (34), 2,4-D (11), dalapon (10), diuron (-2), hexazinone (25), simazine (3), tebuthiuron (21), triclopyr (8), and diesel oil (99).

Routine-realistic applications present high reproductive risks for applicators from atrazine (28), for mixer-loaders from atrazine, and for applicator/mixer-loaders from atrazine. Worst case applications result in reproductive MOSs less than 100 for applicators from atrazine (-5), bromacil (12), clopyralid (94), 2,4-D (31), dalapon (9), dicamba (11), diuron (3), glyphosate (38), hexazinone (70), simazine (2), tebuthiuron (5), and triclopyr (19); for mixer-loaders from atrazine (-2), bromacil (28), 2,4-D (76), dalapon (21), dicamba (27), diuron (7), glyphosate (91), simazine (5), tebuthiuron (11), and triclopyr (45); and for applicator/mixer-loaders from atrazine (-3), bromacil (21), 2,4-D (56), dalapon (15), dicamba (20), diuron (5), glyphosate (67), simazine (3), tebuthiuron (8), and triclopyr (34).

Cancer risks exceed 1 in 1 million for ground mechanical oil and gas site applications for applicators from amitrole (1.78 in 1 million), atrazine (1.09 in 10 thousand), 2,4-D (1.01 in 1 million), and simazine (4.11 in 100 thousand); for mixer-loaders from amitrole (1.05 in 1 million), atrazine (5.75 in 100 thousand), and simazine (2.17 in 100

thousand); and for applicator/mixer-loaders from amitrole (1.28 in 1 million), atrazine (7.21 in 100 thousand), and simazine (2.72 in 100 thousand).

Hand Applications. Systemic, reproductive, and cancer risk estimates for workers in hand oil and gas site applications do not exceed the risk criteria as a result of applying clopyralid, hexazinone, imazapyr, picloram, and kerosene.

Hand herbicide application on oil and gas sites may result in high systemic risk to applicators from the use of atrazine (15), 2,4-D (65), diuron (24), mefluidide (58), metsulfuron methyl (49), sulfometuron methyl (97), or triclopyr (97) in the routine-realistic case. In the worst case, hand applicators have a significant systemic risk from amitrole (35), atrazine (3), bromacil (44), chlorsulfuron (35), 2,4-D (12), dalapon (19), diuron (4), mefluidide (10), metsulfuron methyl (9), simazine (35), sulfometuron methyl (17), tebuthiuron (29), triclopyr (17), and diesel oil (52).

Routine-realistic reproductive MOSs are less than 100 for atrazine (19) and tebuthiuron (65). In the worst case, there are notable reproductive risks from atrazine (3), bromacil (87), 2,4-D (58), dalapon (30), dicamba (42), diuron (44), glyphosate (93), simazine (35), tebuthiuron (12), and triclopyr (70).

Cancer risks to the hand applicator treating oil and gas sites are high from atrazine (2.26 in 100 thousand), 2,4-D (1.79 in 1 million), and simazine (8.52 in 1 million).

Risks From Accidents

Systemic risks from a spill of herbicide concentrate or mixture on the skin are significant for all herbicides used on oil and gas sites. Direct spray of a person poses a high systemic risk from amitrole, atrazine, bromacil, clopyralid, 2,4-D, dalapon, dicamba, diuron, hexazinone, simazine, tebuthiuron, triclopyr, and diesel oil. Immediate reentry by a hiker has a high systemic risk from atrazine and diuron. Drinking water that has been contaminated by a batch truck spill of herbicide mixture intended for treating oil and gas sites results in significant systemic risk from all herbicides except imazapyr.

Reproductive risks from a spill of herbicide concentrate or mixture are significant for all herbicides. Direct spray of a person poses a significant reproductive risk from atrazine, bromacil, 2,4-D, dalapon, dicamba, diuron, glyphosate, simazine, tebuthiuron, and triclopyr. Immediate reentry by a hiker leads

to a significant risk of reproductive effects from atrazine. Drinking water that has been contaminated by a batch truck spill of herbicide mixture poses significant reproductive risks from all herbicides except chlorsulfuron, imazapyr, mefluidide, metsulfuron methyl, diesel oil, and kerosene.

Significant cancer risks are expected from amitrole, atrazine, bromacil, 2,4-D, and simazine as a result of a spill on the skin of herbicide concentrate or mixture or drinking water that has been contaminated by a batch truck spill of herbicide mixture. Direct spray of a person results in a significant cancer risk from atrazine and simazine. No elevated risk of cancer from immediate reentry by a hiker is expected.

Rights-of-Way

MOSs that are less than 100 and cancer risks that are greater than 1 in 1 million as a result of herbicide applications on rights-of-way are presented in Table E5-12 for members of the public, Table E5-13 for workers, and Table E5-14 for accidents. Herbicides used on rights-of-way are amitrole, atrazine, bromacil, chlorsulfuron, clopyralid, 2,4-D, dalapon, dicamba, diuron, glyphosate, hexazinone, imazapyr, mefluidide, metsulfuron methyl, picloram, simazine, sulfometuron methyl, tebuthiuron, triclopyr, and the carriers diesel oil and kerosene.

Risks to Members of the Public

Aerial Applications. For routine-realistic aerial applications on BLM-managed rights-of-way, risks of systemic effects for members of the public are significant for eating fish from a body of water contaminated with amitrole spray drift (38) and the multiple exposures that an angler may receive from amitrole (32). In the routine-worst case, there are high risks due to dermal exposure to spray drift from atrazine (25) and diuron (52); the vegetation contact of a berrypicker from atrazine (10), and diuron (20); drinking water that has received spray drift from amitrole (7), atrazine (25), and diuron (52); eating berries contaminated with drift from amitrole (22) and atrazine (82); eating fish from a body of water contaminated with spray drift from amitrole (1), atrazine (25), diuron (13), and simazine (67); the multiple exposures a hiker may receive from amitrole (7), atrazine (13), and diuron (26); the multiple exposures a berrypicker may receive from amitrole (5), atrazine (5), diuron (11), and simazine (68); the multiple exposures an angler may receive from amitrole (1), atrazine (8), 2,4-D (97), diuron (8), and simazine (46);

and the multiple exposures a nearby resident may receive from atrazine (25) and diuron (51).

Reproductive risk estimates result in MOSs greater than 100 for all herbicides in the routine-realistic case. In the routine-worst case, significant risks are expected for dermal exposure to spray drift from atrazine (33); vegetation contact by a berrypicker from atrazine (13); drinking water that has been contaminated with spray drift from atrazine (33); eating fish from a body of water that has received spray drift from atrazine (33), and simazine (67); the multiple exposures a hiker may have to atrazine (16); the multiple exposures a berrypicker may have to atrazine (7), and simazine (68); the multiple exposures an angler may have to atrazine (10), diuron (83), and simazine (46); and the multiple exposures of a nearby resident to atrazine (33).

Cancer risks are significant for eating fish from a body of water that has been contaminated with amitrole spray drift (1.7 in 1 million) and the multiple exposures that an angler may receive from amitrole (2.11 in 1 million).

Backpack Applications. Risks of systemic effects to members of the public from backpack applications on rights-of-way all have MOSs greater than 100 in the routine-realistic case. In the routine-worst case, there are significant systemic risks for a berrypicker from vegetation contact from atrazine (14) and diuron (28), and for the multiple exposures of a berrypicker from atrazine (14) and diuron (28).

There are no significant reproductive risks to members of the public from routine-realistic backpack applications on rights-of-way. From routine-worst case applications, there is expected to be a significant risk from vegetation contact for a berrypicker from atrazine (18) and multiple exposures to a berrypicker from atrazine (18).

No cancer risk estimates for members of the public exceed a cancer risk of 1 in 1 million for backpack herbicide applications on rights-of-way.

Ground Mechanical Applications. The routine-realistic dose estimated for vegetation contact by a berrypicker results in a significant risk of systemic effects from amitrole (28), as do the multiple exposures received by a berrypicker (28). In the routine-worst case, there is a significant risk of systemic effects from vegetation contact by a berrypicker from

amitrole (2), atrazine (6), diuron (13), and simazine (81); eating fish from a body of water that has been contaminated with amitrole spray drift (44); multiple exposures to a berrypicker from amitrole (2), atrazine (6), diuron (12), and simazine (76); and the multiple exposures an angler may have from amitrole (36).

Routine-realistic exposures are not expected to result in any adverse reproductive effects to members of the public from ground mechanical herbicide applications. However, in the routine-worst case, there are significant reproductive risks from vegetation contact by a berrypicker from atrazine (8), and simazine (81) and from the multiple exposures a berrypicker may receive from atrazine (8), and simazine (76).

Cancer risks exceed 1 in 1 million for vegetation contact by a berrypicker from amitrole (1.76 in 1 million) and for the multiple exposures of a berrypicker to amitrole (1.8 in 1 million).

Risks to Workers

Aerial Applications. MOSs are greater than 100 and cancer risks less than 1 in 1 million for all aerial rights-of-way workers applying chlorsulfuron, imazapyr, metsulfuron methyl, and picloram.

Routine-realistic aerial applications to rights-of-way result in significant systemic risks to pilots from atrazine (96), atrazine (4), diuron (7), and simazine (47) and to mixer-loaders from amitrole (38), atrazine (1), bromacil (59), 2,4-D (63), dalapon (55), diuron (3), simazine (19), and triclopyr (47). There are no high systemic risks in the realistic case to fuel truck operators. In the routine-worst case, there are notable systemic risks to pilots from amitrole (2), atrazine (-13), bromacil (3), clopyralid (10), 2,4-D (3), dalapon (3), dicamba (31), diuron (-6), glyphosate (62), hexazinone (7), mefluidide (48), simazine (-1), sulfometuron methyl (35), tebuthiuron (6), triclopyr (2), and diesel oil (29); to mixer-loaders from amitrole (2), atrazine (-17), bromacil (2), clopyralid (8), 2,4-D (3), dalapon (2), dicamba (25), diuron (-8), glyphosate (48), hexazinone (6), mefluidide (37), simazine (-1), sulfometuron methyl (28), tebuthiuron (5), triclopyr (2), diesel oil (23), and kerosene (87); and to fuel truck operators from amitrole (65), atrazine (2), dalapon (94), diuron (5), simazine (32), and triclopyr (81).

Reproductive risks in the realistic case are significant for pilots from atrazine (5), diuron (74), and simazine (47) and for mixer-loaders

from atrazine (2), dalapon (86), diuron (30), simazine (19), and tebuthiuron (47). There are no significant reproductive risks to fuel truck operators in the realistic case. In the worst case, there are high reproductive risks to pilots from atrazine (-10), bromacil (6), clopyralid (50), 2,4-D (17), dalapon (5), dicamba (6), diuron (2), glyphosate (20), hexazinone (37), simazine (-1), tebuthiuron (2), and triclopyr (10); to mixer-loaders from atrazine (-13), bromacil (5), clopyralid (39), 2,4-D (13), dalapon (4), dicamba (5), diuron (1), glyphosate (16), hexazinone (29), simazine (-1), tebuthiuron (2), and triclopyr (8); and to fuel truck operators from atrazine (3), diuron (50), simazine (32), and tebuthiuron (81).

There are significant cancer risks for pilots from amitrole (4.86 in 1 million), atrazine (3.08 in 10 thousand), bromacil (2.13 in 1 million), 2,4-D (2.44 in 1 million), and simazine (1.16 in 10 thousand); for mixer-loaders from amitrole (7.14 in 1 million), atrazine (4.53 in 10 thousand), bromacil (3.13 in 1 million), and 2, 4-D (3.58 in 1 million), and simazine (1.71 in 10 thousand); and for fuel truck operators from atrazine (9.59 in 1 million) and simazine (3.62 in 1 million).

Backpack Applications. Risk estimates for backpack applicators on rights-of-way do not exceed the systemic, reproductive, or cancer risk criteria as a result of the use of chlorsulfuron, imazapyr, mefluidide, metsulfuron methyl, picloram, or kerosene.

Backpack applicators receiving routine-realistic exposures on rights-of-way are expected to have significant systemic risks from atrazine (26), and diuron (43). In the worst case, high risks result from use of amitrole (23), atrazine (2), bromacil (14), clopyralid (23), 2,4-D (8), dalapon (36), diuron (3), hexazinone (91), simazine (23), sulfometuron methyl (81), triclopyr (11), and diesel oil (67).

Excess reproductive risks to backpack applicators on rights-of-way may result from atrazine (35) under realistic conditions. In the worst case, there may be high reproductive risks from atrazine (2), bromacil (28), 2,4-D (38), dalapon (57), dicamba (27), diuron (28), glyphosate (45), simazine (23), tebuthiuron (61), and triclopyr (45).

There are significant cancer risks to backpack applicators treating rights-of-way with atrazine (1.51 in 100 thousand), 2,4-D (1.19 in 1 million), and simazine (5.69 in 1 million).

Ground Mechanical Applications. MOSs are all greater than 100 and cancer risks less than

1 in 1 million for ground mechanical workers on rights-of-way for applications of chlorsulfuron, imazapyr, metsulfuron methyl, picloram, and kerosene.

Routine-realistic ground mechanical applications of herbicides on rights-of-way may lead to significant systemic risks to applicators from atrazine (43) and diuron (70); to mixer-loaders from atrazine (18), 2,4-D (79), and diuron (29); and to applicator/mixer-loaders from atrazine (18), 2,4-D (81), and diuron (30). Worst case applications may cause high systemic risks to applicators from amitrole (4), atrazine (-7), bromacil (6), clopyralid (19), 2,4-D (4), dalapon (5), dicamba (60), diuron (-3), hexazinone (14), mefluidide (90), simazine (2), sulfometuron methyl (67), tebuthiuron (12), triclopyr (5), and diesel oil (56); to mixer-loaders from amitrole (9), atrazine (-3), bromacil (14), clopyralid (45), 2,4-D (15), dalapon (13), diuron (-1), hexazinone (34), simazine (5), tebuthiuron (28), and triclopyr (11); and for applicator/mixer-loaders from amitrole (7), atrazine (-4), bromacil (10), clopyralid (34), 2,4-D (11), dalapon (10), diuron (-2), hexazinone (25), simazine (3), tebuthiuron (21), triclopyr (8), and diesel oil (99).

In the routine-realistic case, significant reproductive risks are posed for applicators from atrazine (56), for mixer-loaders from atrazine (24), and for applicator/mixer-loaders from atrazine (24). In the worst case, there are notable reproductive risks for applicators from atrazine (-5), bromacil (12), clopyralid (94), 2,4-D (31), dalapon (9), dicamba (11), diuron (3), glyphosate (38), hexazinone (70), simazine (2), tebuthiuron (5), and triclopyr (19); for mixer-loaders from atrazine (-2), bromacil (28), 2,4-D (76), dalapon (21), dicamba (27), diuron (7), glyphosate (91), simazine (5), tebuthiuron (11), and triclopyr (45); and to applicator/mixer-loaders from atrazine (-3), bromacil (21), 2,4-D (56), dalapon (15), dicamba (20), diuron (5), glyphosate (62), simazine (3), tebuthiuron (8), and triclopyr (34).

Significant cancer risks are present for applicators from amitrole (1.72 in 1 million), atrazine (1.06 in 10 thousand), 2,4-D (1.06 in 1 million), and simazine (3.99 in 100 thousand); for mixer-loaders from atrazine (5.01 in 100 thousand) and simazine (1.89 in 100 thousand); and for applicator/mixer-loaders from amitrole (1.14 in 1 million), atrazine (6.49 in 100 thousand), 2,4-D (1.03 in 1 million), and simazine (2.45 in 100 thousand).

Hand Applications. There are no excessive systemic, reproductive, or cancer risks to hand applicators from the use of clopyralid, hexazinone, imazapyr, picloram, or kerosene on rights-of-way.

Workers applying herbicides by hand equipment on rights-of-way are at systemic risk from atrazine (15), 2,4-D (65), diuron (24), mefluidide (58), metsulfuron methyl (49), sulfometuron methyl (97), and triclopyr (97) in the routine-realistic case. Under worst case assumptions, applicators are at high systemic risk from amitrole (35), atrazine (3), bromacil (44), chlorsulfuron (45), 2,4-D (12), dalapon (19), diuron (4), mefluidide (10), metsulfuron methyl (9), simazine (35), sulfometuron methyl (17), tebuthiuron (29), triclopyr (17), and diesel oil (52).

Realistic exposures may result in excess reproductive risks from atrazine (19) and tebuthiuron (65). Worst case exposures may lead to significant reproductive risks from atrazine (3), bromacil (87), 2,4-D (58), dalapon (30), dicamba (42), diuron (44), glyphosate (93), simazine (35), tebuthiuron (12), and triclopyr (70).

Cancer risks to hand applicators on rights-of-way exceed 1 in 1 million for atrazine (2.26 in 100 thousand), 2,4-D (1.79 in 1 million), and simazine (8.52 in 1 million).

Risks From Accidents

There are significant risks of systemic effects from all herbicides used on rights-of-way as a result of a spill onto the skin of herbicide concentrate or mixture. There is a significant risk of systemic effects from a direct spray of a person from all herbicides except chlorsulfuron, glyphosate, imazapyr, mefluidide, metsulfuron methyl, picloram, sulfometuron methyl, and kerosene. There is a significant risk of systemic effects from drinking water that has been directly sprayed with amitrole, atrazine, 2,4-D, dalapon, diuron, simazine, or triclopyr. There is a high systemic risk from eating fish from a body of water that has been directly sprayed with amitrole, atrazine, bromacil, clopyralid, 2,4-D, dalapon, diuron, hexazinone, simazine, and triclopyr. Immediate reentry by a hiker results in significant systemic risks from atrazine and diuron. Immediate reentry by a berrypicker results in significant systemic risks from all herbicides used on rights-of-way except chlorsulfuron, imazapyr, metsulfuron methyl, picloram, and kerosene. Eating directly sprayed berries results in high risks from amitrole, atrazine, bromacil, clopyralid, 2,4-D,

dalapon, diuron, hexazinone, simazine, tebuthiuron, and triclopyr. Drinking water that has been contaminated by a helicopter jettison of herbicide mixture results in high systemic risks from all herbicides except chlorsulfuron, glyphosate, imazapyr, mefluidide, metsulfuron methyl, and kerosene. Drinking water that has been contaminated by a batch truck spill poses significant systemic risks from all herbicides except imazapyr.

There are significant risks of reproductive effects from all herbicides used on rights-of-way as a result of a spill onto the skin of herbicide concentrate or mixture. There is a significant risk of reproductive effects from a direct spray of a person from atrazine, bromacil, 2,4-D, dalapon, dicamba, diuron, glyphosate, simazine, tebuthiuron, and triclopyr. There is a significant risk of reproductive effects from drinking water that has been directly sprayed with atrazine, diuron, simazine, or tebuthiuron. There is a high reproductive risk from eating fish from a body of water that has been directly sprayed with amitrole, atrazine, bromacil, 2,4-D, dalapon, dicamba, diuron, simazine, tebuthiuron, and triclopyr. Immediate reentry by a hiker results in a significant reproductive risk from atrazine. Immediate reentry by a berrypicker results in significant reproductive risks from atrazine, bromacil, clopyralid, 2,4-D, dalapon, dicamba, diuron, glyphosate, hexazinone, simazine, tebuthiuron, and triclopyr. Eating directly sprayed berries results in high risks from amitrole, atrazine, bromacil, 2,4-D, dalapon, dicamba, diuron, simazine, tebuthiuron, and triclopyr. Drinking water that has been contaminated by a helicopter jettison of herbicide mixture results in high reproductive risks from amitrole, atrazine, bromacil, 2,4-D, dalapon, dicamba, diuron, glyphosate, hexazinone, simazine, tebuthiuron, and triclopyr. Drinking water that has been contaminated by a batch truck spill poses significant systemic risks from all herbicides except chlorsulfuron, imazapyr, mefluidide, metsulfuron methyl, and the carriers diesel oil and kerosene.

Significant cancer risks from accidents on rights-of-way are presented by amitrole, atrazine, bromacil, 2,4-D, and simazine for a spill onto the skin of herbicide concentrate or mixture and for drinking water that has been contaminated by a batch truck spill. Amitrole, atrazine, and simazine pose significant cancer risks from eating fish from directly sprayed water, eating berries that have been directly sprayed, or drinking water that has been contaminated by a helicopter jettison of herbicide mixture. Atrazine and simazine pose

a significant cancer risk as a result of a direct spray of a person or immediate reentry to a treated area by a berrypicker. Amitrole and atrazine have a high risk from drinking water that has been directly sprayed. There are no cancer risks greater than 1 in 1 million as a result of immediate reentry by a hiker.

Public Recreation and Cultural Sites

Risks from herbicide applications on public recreation and cultural sites are summarized in Table E5-15 for members of the public, Table E5-16 for workers, and Table E5-17 for accidents. The herbicides used on public recreation and cultural sites are atrazine, chlorsulfuron, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, imazapyr, picloram, simazine, tebuthiuron, triclopyr, and the carriers diesel oil and kerosene.

Risks to Members of the Public

Aerial Applications. BLM does not use aerial applications on public recreation and cultural sites.

Backpack Applications. No significant systemic, reproductive, or cancer risks to members of the public are expected from backpack application of herbicides on BLM-managed public recreation and cultural sites.

Ground Mechanical Applications. No significant systemic, reproductive, or cancer risks to members of the public are expected from ground mechanical application of herbicides on BLM-managed public recreation and cultural sites.

Hand Applications. No significant systemic, reproductive, or cancer risks to members of the public are expected from hand application of herbicides on BLM-managed public recreation and cultural sites.

Risks to Workers

Aerial Applications. Aerial applications are not used on BLM-managed public recreation and cultural sites.

Backpack Applications. There are no significant risks to backpack applicators on BLM-managed public recreation and cultural sites from the use of chlorsulfuron, imazapyr, picloram, tebuthiuron, and kerosene.

Systemic MOSs are greater than 100 for all herbicides in the routine-realistic case. Under worst case assumptions, there are significant systemic risks from atrazine (7), 2,4-D (10),

dalapon (36), hexazinone (91), simazine (91), triclopyr (30), and diesel oil (67).

Reproductive MOSs are greater than 100 for all herbicides in the routine-realistic case. Under worst case assumptions, there are significant reproductive risks from atrazine (9), 2,4-D (50), dalapon (57), dicamba (27), glyphosate (45), and simazine (91).

Cancer risks for backpack applicators exceed 1 in 1 million for atrazine (3.77 in 1 million) and simazine (1.42 in 1 million).

Ground Mechanical Applications. Use of ground mechanical applications on BLM-managed public recreation and cultural sites is not expected to result in significant systemic, reproductive, or cancer risks to workers from the use of chlorsulfuron, hexazinone, imazapyr, picloram, diesel oil, or kerosene.

Systemic MOSs are greater than 100 for all herbicides in the routine-realistic case. Under worst case assumptions, there are significant risks of systemic effects for applicators from atrazine (14), 2,4-D (21), dalapon (75), simazine (47), and triclopyr (63); to mixer-loaders from atrazine (34) and 2,4-D (50); and to applicator/mixer-loaders from atrazine (26), 2,4-D (37), and simazine (84).

Reproductive MOSs are greater than 100 for all herbicides in the routine-realistic case. Under worst case assumptions, there are significant risks of systemic effects for applicators from atrazine (19), dicamba (28), glyphosate (75), simazine (47), and tebuthiuron (47); to mixer-loaders from atrazine (45) and dicamba (68); and to applicator/mixer-loaders from atrazine (34), dicamba (50), simazine (84), and tebuthiuron (84).

Cancer risks exceed 1 in 1 million for applicators from atrazine (1.34 in 1 million) and simazine (1.67 in 1 million), for mixer-loaders from atrazine (1.17 in 1 million), and for applicator/mixer-loaders from atrazine (1.3 in 1 million) and simazine (1.14 in 1 million).

Hand Applications. MOSs are greater than 100 and cancer risks less than 1 in 1 million for hand application workers on public recreation and cultural sites from the use of hexazinone, imazapyr, picloram, and kerosene.

Routine-realistic hand equipment applications may lead to significant systemic risks for applicators from atrazine (15), 2,4-D (65), and triclopyr (97). Worst case applications are estimated to result in systemic risk from atrazine (3), chlorsulfuron (35), 2,4-D (12),

dalapon (19), simazine (35), tebuthiuron (29), triclopyr (17), and diesel oil (52).

Routine-realistic reproductive risks for hand applicators are significant from atrazine (19) and tebuthiuron (65). In the worst case, high risks result from atrazine (3), 2,4-D (58), dalapon (30), dicamba (42), glyphosate (93), simazine (35), tebuthiuron (12), and triclopyr (70).

Excess cancer risks are predicted to result from the use of atrazine (2.26 in 100 thousand), 2,4-D (1.79 in 1 million), and simazine (8.52 in 1 million).

Risks From Accidents

Systemic MOSs for a spill of herbicide concentrate or mixture are less than 100 for all herbicides used on public recreation and cultural sites. Immediate reentry by a hiker would not result in any high systemic risks. Immediate reentry by a berrypicker would pose significant systemic risks from atrazine, 2,4-D, dalapon, dicamba, glyphosate, hexazinone, simazine, tebuthiuron, triclopyr, and diesel oil. Eating directly sprayed berries would result in a high systemic risk from atrazine, 2,4-D, and simazine. Drinking water that was contaminated by a batch truck spill would present significant risks from all herbicides used on recreation and cultural sites except imazapyr.

Reproductive MOSs are less than 100 for all herbicides used on public recreation and cultural sites for a spill of concentrate or mixture on the skin. Immediate reentry by a hiker does not pose any significant reproductive risks. Immediate reentry by a berrypicker presents significant reproductive risks from atrazine, 2,4-D, dalapon, dicamba, glyphosate, simazine, tebuthiuron, and triclopyr. Eating berries that have been directly sprayed results in a high reproductive risk from atrazine, dicamba, simazine, and tebuthiuron. Drinking water that has been contaminated by a batch truck spill of mixture results in significant reproductive risks from all chemicals used on recreation and cultural sites except chlorsulfuron, imazapyr, diesel oil, and kerosene.

Significant cancer risks from accidents on rights-of-way are presented by atrazine, 2,4-D, and simazine for a spill onto the skin of herbicide concentrate or mixture. No significant cancer risks are expected from immediate reentry to a treated area by a hiker or eating directly sprayed berries. Simazine presents a high cancer risk to a berrypicker

that immediately reenters a treated area. Atrazine, 2,4-D, and simazine present significant cancer risks from drinking water that has been contaminated by a batch truck spill of herbicide mixture.

Risk Analysis of Other Effects

This section includes a discussion of risks other than those described under systemic and reproductive effects and cancer risk. This includes risks from inert ingredients in the formulations, risk of heritable mutations, synergistic effects, risks to sensitive individuals, and cumulative effects. This section also includes a discussion on the use of protective clothing, which can reduce worker exposure (and therefore risks).

Risks From Inert Ingredients

In addition to the active ingredient for which risks have been quantified above, pesticide formulations often contain one or more ingredients, such as emulsifiers or surfactants, that aid in ensuring effective application but that are not intended to contribute to the mode of action of toxicity of the mixture. Formulated products are not normally tested in chronic studies, so that kind of information can be considered a data gap in this analysis.

Risk of Heritable Mutations

No human studies are available that associate any of the herbicides with heritable mutations. Furthermore, no risk assessments that quantify the probability of genetic mutations in germ cells are available in the literature or from the Environmental Protection Agency. Laboratory studies constitute the best available information on mutagenic potential. Results of the mutagenicity assays conducted on the herbicides are reported in Section E3, the hazard analysis.

For some of the herbicides, no validated mutagenicity tests exist, or the mutagenicity tests conducted are insufficient to conclude whether the chemical may be mutagenic. For these, a conservative assumption may be made that they have the potential to cause mutations in humans. In these cases, the results of carcinogenicity tests or cancer risk assessments can be used to estimate the risk of heritable mutations.

The rationale for this assumption is summarized by the U.S. Department of Agriculture (1985) as follows:

Since mutagenicity and carcinogenicity both follow similar mechanistic steps (at least those that involve genetic toxicity), the increased risk of cancer can be used to approximate the quantitative risk of heritable mutations. The basis for this assumption is that both mutagens and at least primary carcinogens react with DNA to form a mutation or DNA lesion affecting a particular gene or set of genes. The genetic lesions then require specific metabolic processes to occur, or the cells must divide to insert the lesion into the genetic code of the cell. We believe the cancer risk provides an extreme approximation to heritable mutations because cancer may involve many types of cells, whereas heritable mutations involve only germinal (reproductive) cells.

Therefore, it is conservatively assumed in this risk assessment that the carcinogenic risk estimated for a given exposure also approximates the potential for that herbicide to cause heritable genetic mutations.

Synergistic Effects

Synergistic effects of chemicals are those that occur from exposure to two chemicals either simultaneously or within a relatively short period of time. For example, forestry workers exposed to the fungicide thiram have experienced skin blotching and nausea from drinking alcoholic beverages within 10 days of their thiram exposure. Synergism occurs either when the combined effects of the two chemicals cannot be predicted, based on the known toxic effects of the individual chemicals, or when their combined effect is much greater than the sum of the effects of either chemical given alone. For example, a mixture of the herbicides 2,4-D and picloram has produced skin irritation in test animals, while neither herbicide alone has been found to be a skin irritant. Cigarette smoke and asbestos are both known carcinogens. When inhaled in combination, they have been found to increase cancer risk eightfold above the risk of persons exposed to asbestos who do not smoke.

Instances of chemical combinations that cause synergistic effects are relatively rare. Kociba and Mullison (1985), in describing toxicological interactions with agricultural chemicals, state the following:

Our present scientific knowledge in toxicology indicates than an exposure to a mixture of pesticides is more likely to lead to additivity or antagonism rather than synergism when considering the toxicological effects of such a combination.

To be conservative and for reasons of safety, an additive type of toxicological response is generally assumed rather than an antagonistic type of response.

In the case of registered pesticides, a great amount of toxicological information is developed during the research and development of each individual pesticide. In addition to this information on individual pesticides, short-term toxicity studies are always done before the selling of a pesticide mixture. Should synergism unexpectedly be present in a proposed commercial mixture of two pesticides, it would be identified in such cases and would then be dealt with accordingly. In toxicological tests involving a combination of commercial pesticides, synergism has generally not been observed.

All herbicide mixtures proposed for use in the BLM Vegetation Treatment Program have been approved for use as mixtures by EPA.

The toxic effects of the possible chemical combinations other than the EPA-registered commercial mixtures mentioned are difficult to predict. Time and money normally limit toxicity testing to the effects of individual pesticides. Moreover, the combinations that could be tested are too numerous to make that testing feasible. (The combinations of interest in this risk assessment include not only combinations of 2 or more of the 19 herbicides, but also combinations of the herbicides with other chemicals that may exist in the environment.)

Very little information was available on possible toxic interactions of these herbicides and carriers. Atrazine facilitates the uptake of arsenic by grapes (HSDB 1989). Picloram and 2,4-D have been demonstrated to have a synergistic toxic effect in sheep (HSDB 1989). The toxic effects of kerosene in humans may be exacerbated by administration of antiemetics, epinephrine, or alcohol (HSDB 1989).

The EPA guidelines for assessing the risk from exposures to chemical mixtures (EPA 1986) recommend using additivity models when little information exists on the toxicity of the mixture and when components of the mixture appear to induce the same toxic effect by the same mode of action. They suggest in their discussion of interactions (synergistic or antagonistic effects) of chemical mixtures that "there seems to be a consensus that for public health concerns regarding causative (toxic) agents, the additive model is more appropriate [than any multiplicative model]."

The EPA guidelines suggest using a hazard index (HI) as the model of additivity based on the dose and toxicity reference level (NOEL) for each chemical as follows:

$$HI = D_1/L_1 + D_2/L_2$$

where:

D_1 is the dose of the first component and

L_1 is the toxicity reference level (NOEL)

As HI approaches 1, the risk from the mixture becomes greater.

Effects on Sensitive Individuals

Individual Sensitivity

Doull et al. (1980) describe hypersensitivity as the response of subjects at the lower end of the frequency distribution in a quantal dose-response curve. Quantal means a subject either exhibits the toxic response or does not, at a given dose level. If the response of a population of test animals to varying doses of a chemical follows a normal distribution (bell-shaped curve), the hypersensitive individuals are those on the left hand side of the curve that respond at much lower doses than the average. For example, if the average individual responds with toxic symptoms at a dose of 100 mg/kg and the standard deviation of the response is 30 mg/kg, then about 95 percent of the individuals will have responded with those symptoms at doses from 40 to 160 mg/kg (2 standard deviations from the mean), and more than 99 percent of the individuals will have responded with those symptoms at doses from 10 to 190 mg/kg (3 standard deviations from the mean). Less than 0.15 percent of the population will have experienced toxicity at doses lower than 10 mg/kg. Applying this distribution of response to humans would mean that in a population of 10,000, fewer than 15 individuals would be likely to experience toxicity at doses lower than 10 mg/kg. Those 15 individuals could be considered the sensitive individuals in the population.

Although a safety factor of 10 traditionally has been used by regulatory agencies (NRC 1977) to account for intraspecies (that is, interindividual) variation, Calabrese (1985) has shown that human susceptibility to toxic substances can vary two to three orders of magnitude. Calabrese examined a number of studies of human responses to chemicals and found that the safety factor of 10 accounts for effects in 80 to 95 percent of a population.

Thus, 5 to 20 percent of the population exhibit effects at doses outside the tenfold range.

Factors Affecting the Sensitivity of Individuals

Factors that may affect individual susceptibility to toxic substances include diet, age, heredity, preexisting diseases, and lifestyle (Calabrese 1978). These factors have been studied in detail for very few cases, and their significance in controlling the toxicity of the proposed pesticides is not known. However, enough data have been collected on other chemicals to show that these factors can be important.

Elements of the diet known to affect toxicity include vitamins and minerals (Calabrese and Dorsey 1984). For example, the mineral selenium can prevent the destruction of blood-forming tissues by chronic heavy exposure to benzene. Large doses of vitamin C have also been shown to protect animals and humans from toxic effects of chronic benzene exposure. Vitamin A seems to have a preventative effect on cancer induced by chemicals such as benzo(a)pyrene (found in cigarette and wood smoke) and DMBA. This effect has been seen in laboratory animals and human epidemiological studies. The food additives BHT and BHA also may be active in preventing the carcinogenicity of benzo(a)pyrene. Various levels of the B-vitamin riboflavin have also been tested with mixed results. Vitamin C has been shown to prevent nitrites from combining with amines to form nitrosamines, and vitamin E seems to be at least as effective as vitamin C. These vitamins would be likely to prevent formation of N-nitrosoglyphosate if conditions were otherwise favorable for its formation in the human stomach (Calabrese and Dorsey 1984).

Genetic factors are also known in some cases to be important determinants of susceptibility to toxic environmental agents (Calabrese 1984). Susceptibility to irritants and allergic sensitivity vary widely among individuals and are known to be largely dependent on genetic factors. Race has been shown to be a significant factor influencing sensitivity to irritants, and some investigations have indicated that women may be more sensitive than men (Calabrese 1984).

A variety of human genetic conditions have been identified as possibly enhancing susceptibility to environmental agents. For example, persons with beta-thalassemia may be at increased risk when exposed chronically to benzene. However, only one condition, G-6-PD deficiency, has conclusively been demonstrated to cause enhanced susceptibility to industrial pollutants. Several other genetic conditions have been shown to involve defects in the cellular mechanisms for repair of damage to DNA. Persons with these diseases share an increased sensitivity to the effects of ultraviolet light, which can cause cancer. Cells from individuals with at least one of these diseases, xeroderma pigmentosum, are also sensitive to a variety of chemical substances implicated as causative agents of human cancers (Calabrese 1984).

Persons with other types of preexisting medical conditions may also be at increased risk of toxic effects. For example, sensitivity to chemical skin irritants can be expected to be greater for people with a variety of chronic skin ailments. Patients with these conditions may be advised to avoid occupational exposure to irritating chemicals (Shmunes 1980, as cited in Calabrese 1984).

Allergic Hypersensitivity

A particular form of sensitivity reaction to a foreign substance is allergic hypersensitivity. Allergic hypersensitive reactions may be immediate, such as in anaphylaxis reactions to insect bites or penicillin injections; or they may be delayed as in the case of immune responses to tuberculin tests or contact dermatitis caused by poison ivy. The severe, immediate anaphylaxis reactions, which can be fatal if not treated within minutes, are antigen-antibody reactions that require large, complex organic molecules to initiate the sensitivity. The delayed allergic hypersensitive reactions are usually directed against whole cells (bacteria, viruses, fungi) but, as in contact dermatitis, may be induced by lower molecular weight substances, such as the catechols of poison ivy, cosmetics, drugs, or antibiotics (Volk and Wheeler 1983). Benzocaine, neomycin, formaldehyde, nickel, chromium, and thiram are all known to produce these reactions (Marzulli and Maibach 1983).

Likelihood of Effects in Sensitive Individuals

Based on the current state of knowledge, individual susceptibility to the toxic effects of the 19 herbicides cannot be predicted. As discussed above, safety factors have traditionally been used to account for variations in susceptibility among people. The MOS approach used in this risk assessment takes into account much of the variation in human response as discussed earlier by Calabrese (1985). A safety factor of 10 is used for interspecies variation; an additional safety factor of 10 is used for intraspecies variation.

Thus, the normal MOS of 100 for both types of variation is generally considered by toxicologists to be sufficient to ensure that most people should experience no toxic effects. However, sensitive individuals may experience effects even when the MOS is equal to or greater than 100. In particular, in instances in the risk assessment where the MOS is less than 100 for an exposure to a particular herbicide, it is possible that an exposed sensitive individual would experience toxic effects, whereas the average person may not. It must be noted, however, that sensitive individuals compose only a fraction of the population at large and it is therefore unlikely that a sensitive individual would be among those few people who might be exposed in any of the applications conducted by BLM.

Use of Protective Clothing to Limit Exposure

In estimating potential exposures to workers, various assumptions (described in Section E4) were made regarding the use of protective clothing by persons working directly with herbicides. This section describes the effectiveness of different types of clothing in reducing exposure to pesticides, the role of fabric finishes, and laundering clothing that has pesticide residues.

Worker Studies With Protective Clothing

The use of protective clothing can substantially reduce worker doses, and thereby increase their margins of safety. As shown in a number of relevant field studies, protective clothing can reduce worker exposures by 27 to 99 percent. Typical protective clothing often includes long-sleeved shirts or coveralls, gloves, hats, and boots.

Research has demonstrated that protective clothing can substantially reduce worker doses. For example, in right-of-way spraying, doses received by spray gun applicators wearing clean coveralls and gloves were reduced by 68 percent compared to doses without this protection (Libich et al. 1984). During an aerial spraying operation, mixer-loaders wearing protective clothing reduced their exposure by 58 percent compared to the levels observed without precautions (Lavy et al. 1982).

Most exposure for pesticide applicators is dermal, not inhalation (Kolmodin-Hedman et al. 1983). Wolfe (1972) has indicated that in most pesticide applications, more than 97 percent of the total exposure is dermal. Respirator use is of limited effectiveness in

reducing overall doses to workers and may cause discomfort due to sweating and heat (Davies et al. 1982). The hands are the site of the greatest potential pesticide exposure. Rubber gloves are generally quite effective in reducing exposure to hands (Putnam et al. 1983). Based on a review of the field studies, protective clothing was normally found to reduce mechanical and ground worker doses by 68.1 percent.

Research has shown that most protective clothing, even rubber garments that were previously thought to be impermeable (Mansdorf 1986), allows some level of chemical penetration. However, a study by Davies et al. (1982) showed that when orchard workers wore 100-percent cotton coveralls, dermal doses of the pesticide ethion were less than 15 percent of the doses received when they wore their own street clothes. Putnam and co-workers found that nitrofen applicators and mixer-loaders wearing protective clothing reduced their exposures by 94 to 99 percent compared to the doses experienced without protection (Waldron 1985). Although protective clothing generally reduces worker exposures and resulting doses, the degree of protection depends on the application system, the work practices, and the specific pesticide.

Fabric Finishes

Fabric finishes can also affect doses. Several studies (Laughlin et al. 1986, Leonas et al. 1986, and Keaschall et al. 1986) have shown that fluorocarbon-based soil-repellent finishes increase the effectiveness of clothing as a barrier to chemicals. Water-repellent finishes also contribute to the efficiency of protective garments. In addition, the Laughlin and Leonas studies demonstrated that a durable-press finish is undesirable in clothing worn during pesticide use, because it allows increased penetration of some pesticides, notably methyl parathion. Wearing an undergarment layer, such as a tee-shirt, also decreases the chemical dose received.

Laundering Practices

Laundering practices are important in minimizing pesticide exposure. Heavy duty liquid detergents are more effective than powder detergents in removing oil-based chemicals and in cleaning cotton/polyester blends that have a durable-press finish (Raheel 1987). Using a prewash spray also increases chemical removal (Keaschall et al. 1986). In general, it is more difficult to remove organochlorine residues from clothing than those of organophosphates; and

carbamates are easier to remove than either of these types of pesticides (Raheel 1987, Keaschall et al. 1986). Clothes worn during any exposure to pesticides should be washed separately from other clothes, to avoid potential transfer of residues.

Table E5-3. High Risks to Members of the Public From Herbicide Use on Rangeland

Exposure Scenario	Typical Exposures		Worst-case Exposures		
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	—	—	—
Drinking Water	—	—	AM	—	—
Eating Berries	—	—	—	—	—
Eating Fish	AM	—	AM, 4D	—	—
Hiker	—	—	AM	—	—
Berrypicker	—	—	AM	—	—
Angler	AM	—	AM, 4D	—	AM
Nearby Resident	—	—	—	—	—
Backpack Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	—	—	—
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	—	—	—
Hiker	—	—	—	—	—
Berrypicker	—	—	—	—	—
Angler	—	—	—	—	—
Nearby Resident	—	—	—	—	—
Ground Mechanical Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	AM	—	AM	—	AM
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	—	—	—
Hiker	—	—	—	—	—
Berrypicker	AM	—	AM	—	AM
Angler	—	—	—	—	—
Nearby Resident	—	—	—	—	—

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10^{-6}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-4. High Risks to Workers From Herbicide Use on Rangeland

Exposure Scenario	Typical Exposures		Worst-case Exposures		
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Pilot	AM, AT, 4D, TC	AT, 4D, DC, TB	AM, AT, 4D, DP, DC, GP, HX, TB, TC, DE, KE	AT, 4D, DP, DC, GP, TB, TC	AM, AT, 4D
Mixer-loader	AM, AT, 4D, DP, DC, TB, TC, DE	AT, 4D, DP, DC, GP, TB	AM, AT, CP, 4D, DP, DC, GP, HX, TB, TC, DE, KE	AT, 4D, DP, DC, GP, TB, TC	AM, AT, 4D
Fuel Truck Operator	—	—	AT, 4D	AT, DC	—
Backpack Applications					
Applicator	—	—	AM, AT, 4D, DP, TC, DE	AT, 4D, DP, DC, GP	AT, 4D
Ground Mechanical Operations					
Applicator	AT, 4D	AT	AM, AT, 4D, DP, DC, GP, TB, TC, DE	AT, 4D, DP, DC, GP, TB, TC	AT, 4D
Mixer-loader	AM, AT, 4D	AT	AM, AT, 4D, DP, DC, TB, TC, DE	AT, 4D, DP, DC, GP, TB	AT, 4D
Applicator/mixer-loader	AT, 4D	AT	AM, AT, 4D, DP, DC, GP, TB, TC, DE	AT, 4D, DP, DC, GP, TB, TC	AT, 4D

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1×10^{-6}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-5. High Risks From Accidents From Herbicide Use on Rangeland

Exposure Scenario	Systemic	Reproductive	Cancer
Skin Spill, Concentrate	AM, AT, CP, 4D, DC, GP, HX, IP, PC, TC, DE, KE	AM, AT, CP, 4D, DC, GP, HX, IP, PC, TC, DE, KE	AM, AT, 4D
Skin Spill, Mixture	AM, AT, CP, 4D, DP, DC, GP, HX, IP, PC, TB, TC, DE, KE	AM, AT, CP, 4D, DP, DC, GP, HX, IP, PC, TB, TC, DE, KE	AM, AT, 4D
Direct Spray, Person	AM, AT, 4D, DP, DC, TB, TC	AT, 4D, DC, GP, TB, DE	—
Drinking Directly Sprayed Water	AM, 4D	—	—
Eating Fish From Directly Sprayed Water	AM, 4D, TC	4D, DC	AM
Immediate Reentry, Hiker	—	—	—
Immediate Reentry, Picker	AM, AT, 4D, DP, DC GP, TB, TC, DE	AT, 4D, DP, DC, GP, TB, TC	—
Eating Directly Sprayed Berries	AM, AT, 4D	AT, 4D, DC, TB	AM
Drinking Water Contaminated by a Jettison of Mixture	AM, AT, 4D, DP, DC, PC, TB, TC, DE	AM, AT, 4D, DP, DC, GP, TB	AM
Drinking Water Contaminated by a Truck Spill	AM, AT, CP, 4D, DP, DC, GP, HX, PC, TB, TC, DE, KE	AM, AT, 4D, DP, DC, GP, HX, PC, TB, TC	AM, AT, 4D

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron;
GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine;
SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-6. High Risks to Members of the Public From Herbicide Use on Public-Domain Forest Land

Exposure Scenario	Typical Exposures		Worst-case Exposures		
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	AT	—	—
Drinking Water	—	—	AM	—	—
Eating Berries	—	—	—	—	—
Eating Fish	AM	—	AM, 4D	—	—
Hiker	—	—	AM	—	—
Berrypicker	—	—	AM, AT, 4D	AT	—
Angler	AM	—	AM, AT, 4D	—	AM
Nearby Resident	—	—	—	—	—
Backpack Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	—	—	—
Drinking Water	—	—	AM	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	AM, 4D	—	—
Hiker	—	—	AM	—	—
Berrypicker	—	—	AM, AT	AT	—
Angler	—	—	AM, AT, 4D	AT	—
Nearby Resident	—	—	—	—	—
Ground Mechanical Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	AM	—	AM, AT, 4D	AT	AM
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	—	—	—
Hiker	—	—	—	—	—
Berrypicker	AM	—	AM, AT, 4D	AT	AM
Angler	—	—	—	—	—
Nearby Resident	—	—	—	—	—

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate;
HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl;
TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-7. High Risks to Workers From Herbicide Use on Public-Domain Forest Land

Exposure Scenario	Typical Exposures		Worst-case Exposures		
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Pilot	AT, 4D	AT	AM, AT, 4D, DP, DC, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, 4D, SI
Mixer-loader	AT, 4D, TC	AT	AM, AT, 4D, DP, DC, GP, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AM, AT, 4D, SI
Fuel Truck Operator	—	—	AT, 4D	AT	—
Backpack Applications					
Applicator	AT	AT	AM, AT, 4D, DP HX, SI, TC, DE	AT, 4D, DP, DC GP, SI, TB, TC	AT, 4D, SI
Ground Mechanical Operations					
Applicator	AT, 4D	AT	AM, AT, 4D, DP, DC, GP, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, 4D, SI
Mixer-loader	AT, 4D	AT	AM, AT, 4D, DP, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, 4D, SI
Applicator/mixer-loader	AT, 4D	AT	AM, AT, 4D, DP, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, 4D, SI
Hand Applications					
Applicator	AT, 4D, TC	AT, TB	AM, AT, CS, 4D, DP SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁴.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-8. High Risks From Accidents From Herbicide Use on Public-Domain Forest Land

Exposure Scenario	Systemic	Reproductive	Cancer
Skin Spill, Concentrate	AM, AT, CS, 4D, DC, GP, HX, IP, PC, SI, TC, DE, KE	AM, AT, CS, 4D, DC, GP, HX, IP, PC, SI, TC, DE, KE	AM, AT, 4D, SI
Skin Spill, Mixture	AM, AT, CS, 4D, DP, DC, GP, HX, IP, PC, SI, TB, TC, DE, KE	AM, AT, CS, 4D, DP, DC, GP, HX, IP, PC, SI, TB, TC, DE, KE	AM, AT, 4D, SI
Direct Spray, Person	AM, AT, 4D, DP, DE HX, SI, TB, TC	AT, 4D, DP, DC, GP, SI, TB, TC	AT
Drinking Directly Sprayed Water	AM, AT, 4D	AT	—
Eating Fish From Directly Sprayed Water	AM, AT, 4D, SI, TC	AT, 4D, DC, SI	AM
Immediate Reentry, Hiker	—	—	—
Immediate Reentry, Picker	AM, AT, 4D, DP, DC, GP, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, SI
Eating Directly Sprayed Berries	AM, AT, 4D, SI, TC	AT, 4D, DC, SI, TB	AM
Drinking Water Contaminated by a Jettison of Mixture	AM, AT, 4D, DP, DC, HX, PC, SI, TB, TC, DE	AM, AT, 4D, DP, DC, GP, SI, TB, TC	AM, AT
Drinking Water Contaminated by a Truck Spill	AM, AT, CS, 4D, DP, DC, GP, HX, PC, SI, TB, TC, DE, KE	AM, AT, 4D, DP, DC, GP, HX, PC, SI, TB, TC	AM, AT, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-9. High Risks to Members of the Public From Herbicide Use on Oil and Gas Sites

| Exposure Scenario | Typical Exposures | | Worst-case Exposures | | |
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Spray Drift, Dermal	—	—	AT, DU	AT	—
Vegetation Contact, Hiker	—	—	—	—	—
Hiker	—	—	AT, DU	AT	—
Nearby Resident	—	—	AT, DU	AT	—
Backpack Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Hiker	—	—	—	—	—
Nearby Resident	—	—	—	—	—
Ground Mechanical Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Hiker	—	—	—	—	—
Nearby Resident	—	—	—	—	—

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron;
GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine;
SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-10. High Risks to Workers From Herbicide Use on Oil and Gas Sites

| Exposure Scenario | Typical Exposures | | Worst-case Exposures | | |
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Pilot	AM, AT, DU, SI	AT, DU, SI	AM, AT, BR, CP, 4D, DP, DC, DU, HX, SI, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AM, AT, 4D, SI
Mixer-loader	AM, AT, BR, 4D, DP, SI, TC	AT, DP, DU, SI, TB	AM, AT, BR, CP, 4D, DP, DC, DU, HX, SI, SM, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AM, AT, BR, 4D, SI
Fuel Truck Operator	—	—	AT, DU, SI	AT, SI	AT, SI
Backpack Applications					
Applicator	AT, DU	AT	AM, AT, BR, CP, 4D, DP, DC, DU, HX, MF, SI, SM, TB, TC, DE	AT, BR, CP, 4D, DP, DC, DU, GP, SI, TB, TC	AM, AT, 4D, SI
Ground Mechanical Operations					
Applicator	AT, DU	AT	AM, AT, BR, CP, 4D, DP, DC, DU, HX, MF, SI, SM, TB, TC, DE	AT, BR, CP, 4D, DC, DP, DU, GP, HX, SI, TB, TC	AM, AT, 4D, SI
Mixer-loader	AT, 4D, DU	AT	AM, AT, BR, CP, 4D, DP, DU, HX, SI, TB, TC	AT, BR, 4D, DP, DC, DU, SI, TB, TC	AM, AT, SI
Applicator/mixer-loader	AT, DU	AT	AM, AT, BR, CP, 4D, DP, DU, HX, SI, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AM, AT, SI
Hand Applications					
Applicator	AT, 4D, DU, MF, MM, SM, TC	AT, TB	AM, AT, BR, CS, 4D, DP, DU, MF, MM, SI, SM, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AT, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1×10^{-4}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-11. High Risks From Accidents From Herbicide Use on Oil and Gas Sites

Exposure Scenario	Systemic	Reproductive	Cancer
Skin Spill, Concentrate	All except DP, TB	All, except DP, TB	AM, AT, BR, 4D, SI
Skin Spill, Mixture	All	All	AM, AT, BR, 4D, SI
Direct Spray, Person	AM, AT, BR, CP, 4D, DP, DC, DU, HX, SI, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AT, SI
Drinking Directly Sprayed Water	—	—	—
Eating Fish from Directly Sprayed Water	—	—	—
Immediate Reentry, Hiker	AT, DU	AT	—
Immediate Reentry, Picker	—	—	—
Eating Directly Sprayed Berries	—	—	—
Drinking Water Contaminated by a Jettison of Mixture	—	—	—
Drinking Water Contaminated by a Truck Spill	All except imazapyr	AM, AT, BR, CP, 4D, DP, DC, DU, GP, HX, PC, SI, SM, TB, TC	AM, AT, BR, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1×10^{-6}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate;
HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron;
TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-12. High Risks to Members of the Public From Herbicide Use on Rights-of-Way

Exposure Scenario	Typical Exposures		Worst-case Exposures		
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Spray Drift, Dermal	—	—	AT, DU	AT	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	AT, DU	AT	—
Drinking Water	—	—	AM, AT, DU	AT	—
Eating Berries	—	—	AM, AT	—	—
Eating Fish	AM	—	AM, AT, DU, SI	AT, SI	AM
Hiker	—	—	AM, AT, DU	AT	—
Berrypicker	—	—	AM, AT, DU, SI	AT, SI	—
Angler	AM	—	AM, AT, 4D, DU, SI	AT, DU, SI	AM
Nearby Resident	—	—	AT, DU	AT	—
Backpack Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	AT, DU	AT	—
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	—	—	—
Hiker	—	—	—	—	—
Berrypicker	—	—	AT, DU	AT	—
Angler	—	—	—	—	—
Nearby Resident	—	—	—	—	—
Ground Mechanical Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	AM	—	AM, AT, DU, SI	AT, SI	AM
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	AM	—	—
Hiker	—	—	—	—	—
Berrypicker	AM	—	AM, AT, DU, SI	AT, SI	AM
Angler	—	—	AM	—	—
Nearby Resident	—	—	—	—	—

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1×10^{-4}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-13. High Risks to Workers From Herbicide Use on Rights-of-Way

| | Typical Exposures | | Worst-case Exposures | | |
Exposure Scenario	Systemic	Reproductive	Systemic	Reproductive	Cancer
Aerial Applications					
Pilot	AM, AT, DU, SI	AT, DU, SI	AM, AT, BR, CP, 4D, DP, DC, DU, GP, HX, MF, SI, SM, TB, TC, DE	AT, BR, CP, 4D, DP, DC, DU, GP, HX, SI, TB, TC	AM, AT, BR, 4D, SI
Mixer-loader	AM, AT, BR, 4D, DP, DU, SI, TC	AT, DP, DU, SI, TB	All except CS, IP, MM, PC	AT, BR, CP, 4D, DP, DC, DU, GP, HX, SI, TB, TC	AM, AT, BR, 4D, SI
Fuel Truck Operator	—	—	AM, AT, DP, DU, SI, TC	AT, DU, SI, TB	AT, SI
Backpack Applications					
Applicator	AT, DU	AT	AM, AT, BR, CP, 4D, DP, DU, HX, SI, SM, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AT, 4D, SI
Ground Mechanical Operations					
Applicator	AT, DU	AT	AM, AT, BR, CP, 4D, DP, DC, DU, HX, MF, SI, SM, TB, TC, DE	AT, BR, CP, 4D, DP, DC, DU, GP, HX, SI, TB, TC	AM, AT, 4D, SI
Mixer-loader	AT, 4D, DU	AT	AM, AT, BR, CP, 4D, DP, DU, HX, SI, TB, TC	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AT, SI
Applicator/mixer-loader	AT, 4D, DU	AT	AM, AT, BR, CP, 4D, DP, DU, HX, SI, TB, TC, DE	AT, BR, 4D, DP, DC DU, GP, SI, TB, TC	AM, AT, 4D, SI
Hand Applications					
Applicator	AT, 4D, DU, MF, MM, SM, TC	AT, TB	AM, AT, BR, CS, 4D, DP, DU, MF, MM, SI, SM, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, SM, TB, TC	AT, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-14. High Risks From Accidents From Herbicide Use on Rights-of-Way

Exposure Scenario	Systemic	Reproductive	Cancer
Skin Spill, Concentrate	All except DP, TB	All except DP, TB	AM, AT, BR, 4D, SI
Skin Spill, Mixture	All	All	AM, AT, BR, 4D, SI
Direct Spray, Person	AM, AT, BR, CP, 4D, DP, DC, DU, HX, SI, TB, TC, DE	AT, BR, 4D, DP, DC, DU, GP, SI, TB, TC	AT, SI
Drinking Directly Sprayed Water	AM, AT, 4D, DP, DU, SI, TC	AT, DU, SI, TB	AM, AT
Eating Fish from Directly Sprayed Water	AM, AT, BR, CP, 4D, DP, DU, HX, SI, TC	AM, AT, BR, 4D, DP, DC, DU, SI, TB, TC	AM, AT, SI
Immediate Reentry, Hiker	AT, DU	AT	—
Immediate Reentry, Picker	AM, AT, BR, CP, 4D, DP, DC, DU, GP, HX, MF, SI, SM, TB, TC, DE	AT, BR, CP, 4D, DP, DC, DU, GP, HX, SI, TB, TC	AT, SI
Eating Directly Sprayed Berries	AM, AT, BR, CP, 4D, DP, DU, HX, SI, TB, TC	AM, AT, BR, 4D, DP, DC, DU, SI, TB, TC	AM, AT, SI
Drinking Water Contaminated by a Jettison of Mixture	AM, AT, BR, CP, 4D, DP, DC, DU, HX, PC, SI, SM, TB, TC, DE	AM, AT, BR, 4D, DP, DC, DU, GP, HX, SI, TB, TC	AM, AT, SI
Drinking Water Contaminated by a Truck Spill	All except IP	AM, AT, BR, CP, 4D, DP, DC, DU, GP, HX, PC, SI, SM, TB, TC	AM, AT, BR, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-15. High Risks to Members of the Public From Herbicide Use on Recreation and Cultural Sites

| | Typical Exposures | | Worst-case Exposures | | |
Exposure Scenario	Systemic	Reproductive	Systemic	Reproductive	Cancer
Backpack Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	—	—	—
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	—	—	—
Hiker	—	—	—	—	—
Berrypicker	—	—	—	—	—
Angler	—	—	—	—	—
Nearby Resident	—	—	—	—	—
Ground Mechanical Applications					
Spray Drift, Dermal	—	—	—	—	—
Vegetation Contact, Hiker	—	—	—	—	—
Vegetation Contact, Picker	—	—	—	—	—
Drinking Water	—	—	—	—	—
Eating Berries	—	—	—	—	—
Eating Fish	—	—	—	—	—
Hiker	—	—	—	—	—
Berrypicker	—	—	—	—	—
Angler	—	—	—	—	—
Nearby Resident	—	—	—	—	—

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1×10^{-6}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-16. High Risks to Workers From Herbicide Use on Recreation and Cultural Sites

| Exposure Scenario | Typical Exposures | | Worst-case Exposures | | |
	Systemic	Reproductive	Systemic	Reproductive	Cancer
Backpack Applications					
Applicator	—	—	AT, 4D, DP, HX, SI, TC, DE	AT, 4D, DP, DC, GP, SI	AT, SI
Ground Mechanical Operations					
Applicator	—	—	AT, 4D, DP, SI, TC	AT, DC, GP, SI, TB	AT, SI
Mixer-loader	—	—	AT, 4D	AT, DC	AT
Applicator/mixer-loader	—	—	AT, 4D, SI	AT, DC, SI, TB	AT, SI
Hand Applications					
Applicator	AT, 4D, TC	AT, TB	AT, CS, 4D, DP, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	AT, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1 x 10⁻⁶.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

Table E5-17. High Risks From Accidents From Herbicide Use on Recreation and Cultural Sites

Exposure Scenario	Systemic	Reproductive	Cancer
Skin Spill, Concentrate	All except DP, TB	All except DP, TB	AT, 4D, SI
Skin Spill, Mixture	All	All	AT, 4D, SI
Immediate Reentry, Hiker	—	—	—
Immediate Reentry, Picker	AT, 4D, DP, DC, GP, HX, SI, TB, TC, DE	AT, 4D, DP, DC, GP, SI, TB, TC	SI
Eating Directly Sprayed Berries	AT, 4D, SI	AT, DC, SI, TB	—
Drinking Water Contaminated by a Truck Spill	All except IP	AT, 4D, DP, DC, GP, HX, PC, SI, TB, TC	AT, 4D, SI

Note: High risks are defined as those exposures that may result in a margin of safety less than 100 or a cancer risk greater than 1×10^{-6}.

Legend:
AM = Amitrole; AT = Atrazine; BR = Bromacil; CS = Chlorsulfuron; CP = Clopyralid; 4D = 2,4-D; DP = Dalapon; DC = Dicamba; DU = Diuron; GP = Glyphosate; HX = Hexazinone; IP = Imazapyr; MF = Mefluidide; MM = Metsulfuron methyl; PC = Picloram; SI = Simazine; SM = Sulfometuron methyl; TB = Tebuthiuron; TC = Triclopyr; DE = Diesel; KE = Kerosene.

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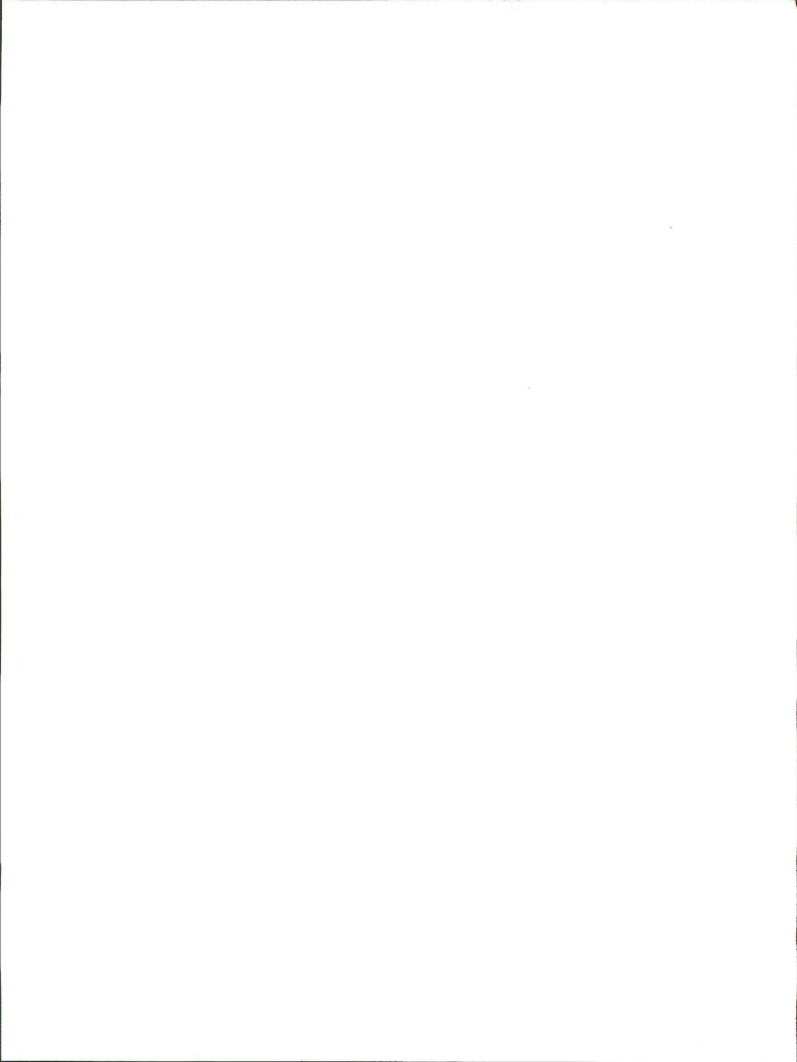
Section E6
Nontarget Species Hazard Analysis

This section summarizes the toxicity to wildlife and aquatic species of the 19 herbicides—amitrole, atrazine, bromacil, chlorsulfuron, clopyralid, 2,4-D, dalapon, dicamba, diuron, glyphosate, hexazinone, imazapyr, mefluidide, metsulfuron methyl, picloram, simazine, sulfometuron methyl, tebuthiuron, and triclopyr—proposed for use for BLM vegetation treatments. The term wildlife as used in this section refers to mammals, birds, reptiles, amphibians, and insects; aquatic species include fish, aquatic invertebrates, and aquatic life-stages of amphibians. Wildlife and aquatic species are discussed in separate subsections, each with an introduction that includes information on toxicity classifications and terminology.

Wildlife Hazard Analysis

This hazard analysis summarizes the findings of laboratory and field studies that indicate the toxicity to wildlife of the herbicides and additives proposed for BLM use for vegetation management. In many cases, laboratory studies of domestic animals have been used because of the lack of specific wildlife studies. The results of domestic animal studies are considered to be representative of the effects that would occur in similar species in the wild.

Differences in sensitivity to toxic substances that occur between species are primarily accounted for by differences in metabolism (Calabrese 1983). Other important factors that also account for these differences in sensitivity are absorption, plasma protein binding, biliary excretion, and intestinal microflora (Calabrese 1983).

Mammalian toxicity studies, as well as carcinogenicity and mutagenicity results, have already been summarized in Section E3, Human Health Hazard Analysis, and will not be repeated in detail here. The relative toxicity of the chemicals, based on the range of LD_{50} values, was based on the same toxicity categories EPA used for humans (see Section E3). The toxicity rating used in this risk assessment for honey bees is that of Dr. Larry Atkins (University of California). It is based on the amount of herbicide required to kill a bee: less than 2 micrograms (µg)/bee is classified as highly toxic, 2 to 11 µg/bee is

moderately toxic, and greater than 11 µg/bee is relatively nontoxic (Vaughan, personal communication 1987).

The acute toxicity of the BLM herbicides and additives to rats and mallards is summarized in Table E6-1.

Amitrole

Amitrole is slightly toxic to birds and mammals based on acute oral LD_{50}s ranging from greater than 1,100 mg/kg in rats to greater than 14,700 mg/kg in mice (Table E6-2). The acute oral LD_{50} in mallards is greater than 2,000 mg/kg (Hudson et al. 1984). The 8-day dietary LC_{50} of amitrole in Japanese quail, mallards, and ring-necked pheasants is greater than 5,000 ppm (USDA 1984). Toxic effects in birds include muscle incoordination, weakness, and slight wing-drop (Hudson et al. 1984). No reproductive or teratogenic effects were caused by amitrole in mice or rats (EPA 1984a). However, depressed reproduction was observed in mallards fed dose levels 25 percent below the lethal dose (USDA 1984). Chicken eggs injected with amitrole exhibited toxic effects at 100 ppm in one study and malformations of the beak and tibia at 40 mg/egg in another study (USDA 1984). The LC_{50} of mallard eggs immersed in an aqueous solution of amitrole is a concentration equivalent to a field application rate of 176 lb/acre (Hoffman and Albers 1984). Amitrole is primarily excreted unchanged in the urine and does not appear to bioaccumulate in the tissues of animals (USDA 1984).

Atrazine

Atrazine is of low toxicity to birds and mammals based on acute oral LD_{50}s ranging from 672 mg/kg to 4,237 mg/kg in Japanese quail (Table E6-3) (EPA 1983a, Gaines and Linder 1968, as cited in EPA 1987a). Toxic effects in mammals include sedation, labored breathing, ruffled fur, and protruding eyeballs (EPA 1984b). Atrazine did not cause irritation or sensitization of the skin in rabbits and guinea pigs, but was irritating to the eyes of rabbits (EPA 1984b). In a 28-day feeding study, body weight loss was observed in sheep at the lowest dose tested of 50 mg/kg/day of the Atrazine 80W formulation

Table E6-1. Acute Toxicity of BLM Herbicides and Additives to Rats and Mallard Ducks

Herbicide/Additive	Oral LD$_{50}$ (mg/kg)	
	Rat	Mallard
Amitrole	>1,100	>2,000
Atrazine	672	>2,000
Bromacil	3,998	No data
Chlorsulfuron	5,545	>5,000
Clopyralid	4,300	No data
2,4-D		
Acid	375	>2,000
Butyl Ester	620	>2,025
Dalapon	7,570	No data
Dicamba (Banvel)	757	>2,510
Diuron	3,400	2,000
Glyphosate	4,320	>2,000[a]
Hexazinone	1,690	>1,250[b]
Imazapyr	>5,000	>2,150
Kerosene	>28,000	No data
Diesel Oil	>7,380	16,400
Mefluidide	>4,000	No data
Metsulfuron methyl	>5,000	No data
Picloram	4,012	>2,000
Simazine	>5,000	>4,640
Sulfometuron Methyl	>5,000	>5,000
Tebuthiuron	644	>2,000
Triclopyr, Technical	630	1,698
Garlon 3A (amine)	2,830	No data
Garlon 4 (ester)	2,140	>4,640

[a]Bobwhite; no value for the mallard is available.
[b]Based on a dietary LC$_{50}$ for mallards of 10,000 ppm and a conversion factor of 0.125 mg/kg/day per ppm in diet for chicks (Lehman 1954).

Table E6-2. Acute Oral Toxicity of Amitrole to Birds and Mammals

Species	LD$_{50}$ (mg/kg)
Rat	>1,100
Mouse	>14,700
Rabbit	>10,000
Cat	>1,750
Mallard	>2,000
Chicken	>2,000

Source: EPA 1984a, Hudson et al. 1984, USDA 1984.

Table E6-3. Acute Oral Toxicity of Atrazine to Birds and Mammals

Species	LD$_{50}$ (mg/kg)
Rat	672
Mouse	1,750
Japanese Quail	4,237
Bobwhite Quail	940
Mallard Duck	>2,000
Pheasant	>2,000

Source: EPA 1983a, EPA 1984b, Hudson et al. 1984, USDA 1984; Gaines and Linder 1986, as cited in EPA 1987a.

(EPA 1984b). In another study, no effects were observed in sheep given 25 mg/kg/day for 35 days (EPA 1984b). No adverse effects were observed in ewes treated with 15 mg/kg/day throughout pregnancy (USDA 1984), and no teratogenic or reproductive effects have been reported for mice and rats (EPA 1984b).

Avian toxic effects from atrazine include weakness, hyperexcitability, muscle incoordination, tremors, and weight loss (Hudson et al. 1984). Eight-day dietary LC$_{50}$s for the 99-percent active ingredient were all greater than 5,000 ppm in Japanese quail, bobwhite quail, pheasants, and mallards (EPA 1983a). Pheasants given 15 weekly doses of up to 400 mg of atrazine (80 percent a.i.) showed no change in weight gain, number of eggs laid, eggshell thickness, survival, and weight of offspring (Melius 1975, as cited in USDA 1984). Injection of eggs with atrazine caused reduced hatching at 400 ppm (the highest dose tested) (Dunachie and Fletcher 1970, as cited in USDA 1984). No teratogenic effects were observed. The LC$_{50}$ of mallard eggs immersed in an aqueous emulsion of atrazine is a concentration equivalent to a field application greater than 400 lb/acre (Hoffman and Albers 1984).

Atrazine has a low potential for bioaccumulation in animals and is readily metabolized to nontoxic metabolites and rapidly excreted through the kidneys (USDA 1984).

Bromacil

Bromacil is slightly toxic to birds and mammals based on the acute oral LD$_{50}$ of 3,998 mg/kg in rats (EPA 1984c) and the subacute 8-day dietary LC$_{50}$ of greater than 10,000 ppm (1,750 mg/kg) in bobwhite quail and mallard ducks (EPA 1985a). Bromacil caused mild skin and eye irritation in rabbits (EPA 1984c). No reproductive or teratogenic effects have been observed in rats and rabbits exposed to bromacil (EPA 1984c). Sheep given oral doses of 50 mg/kg for 10 days experienced an 8-percent reduction in body weight (Palmer and Radeleff 1969). Based on these results, application rates of greater than 5 lb/acre are considered hazardous to sheep.

Chickens given 10 daily doses of 250 mg/kg showed a reduced rate of weight gain (Palmer and Radeleff 1969). Application rates of 20 lb/acre are therefore considered hazardous to chickens.

Chlorsulfuron

Chlorsulfuron is of low toxicity to birds and mammals based on acute oral LD$_{50}$s of 5,545 mg/kg in rats and greater than 5,000 mg/kg in bobwhite quail and mallard ducks (WSSA 1983). Mallards and bobwhite quail also have an 8-day dietary LC$_{50}$ of greater than 5,000 ppm/day (Worthing 1983). Toxicity values on other terrestrial wildlife were not reported.

Clopyralid

Clopyralid is of low toxicity to birds and mammals based on the acute oral LD$_{50}$ of greater than 4,300 mg/kg in rats (Agrochemicals Handbook 1988), greater than 5,000 mg/kg in mice (WSSA 1983), and 1,465 mg/kg in ducks (NIOSH 1989) (Table E6-4). Both quail and ducks have 8-day dietary LC$_{50}$s of greater than 4,640 ppm.

Table E6-4. Acute Oral Toxicity of Clopyralid to Birds and Mammals

Species	LD₅₀ (mg/kg)
Rat	>4,300
Duck	1,465
Mouse	>5,000
Bees	>100 µg/bee

Source: WSSA 1983, NIOSH 1989, Agrochemicals Handbook 1988.

Clopyralid is nontoxic to bees. Both the oral 48-hour LD_{50} and contact LD_{50} were greater than 100 µg/bee (Agrochemicals Handbook 1988).

2,4-D

2,4-D is moderately toxic to vertebrate species (Table E6-5). There are significant differences in toxicity to vertebrates among the forms of 2,4-D (amines, butyl esters, isooctyl esters, and propylene glycol butyl ether esters) (Ghassemi et al. 1981). In many instances, toxic response to a specific 2,4-D formulation appears to be species-specific (USDA 1984). Oral LD_{50}s in mammals range from 100 mg/kg for dogs, cattle, and swine to 848 mg/kg for guinea pigs (USDA 1984, Ghassemi et al. 1981). Toxic effects include gastrointestinal disturbances, weight loss, muscle weakness, and loss of coordination (USDA 1984). Mild to moderate eye, skin, and respiratory irritation is caused by some formulations (USDA 1984). No teratogenic or reproductive effects have been observed in rats (EPA 1986a).

In birds, acute oral LD_{50}s range from 472 mg/kg in pheasants (3 to 4 months old) to more than 2,000 mg/kg in mallards (4 months old) (Hudson et al. 1984). Toxic effects include excessive thirst and salivation, tremors, exhaustion, and imbalance (Hudson et al. 1984). Eight-day dietary studies with the dimethylamine salt of 2,4-D and the butoxyethanol ester of 2,4-D yielded LC_{50} values of more than 5,000 ppm for Japanese quail, bobwhite quail, ring-necked pheasants, and mallard ducks (Hill et al. 1975, as cited in USDA 1984). No reproductive or teratogenic effects were observed in the eggs of chickens and pheasants when sprayed with various forms of 2,4-D, even at dosage levels of up to 20 times the recommended field application rate (USDA 1984). Chicken eggs injected with 2,4-D to give concentrations of 10, 50, 100,

200, and 300 ppm in the eggs resulted in hatching success rates of 83, 100, 71, 62, and 0 percent, respectively, of the control hatch (Dunachie and Fletcher 1970, as cited in USDA 1984). The LC_{50} of mallard eggs immersed in an aqueous emulsion of 2,4-D was a concentration equivalent to a field application rate of 215 kg/ha (192 lb/acre) (Hoffman and Albers 1984).

The bioaccumulation ratio is low for tested animals exposed to 2,4-D and accumulated residues are rapidly excreted once exposure ceases (Norris 1981, as cited in USDA 1984). Very few monitoring data exist on 2,4-D levels found in wildlife. However, studies by Erne (1974) in Sweden found levels of 2,4-D residues that ranged from 0.05 to 6 mg/kg in liver and kidney tissue of 250 samples of wildlife (including moose, roedeer, reindeer, red deer, fallow deer, hares, pheasants, grouse, and other species) taken by hunters or found dead during the period 1968 to 1972.

There is some indication in the literature that after treatment with 2,4-D, palatability (and possibly increased toxicity) of normally unpalatable weeds increases (Irvine et al. 1977). This was observed in ragwort (*Senecio jacobaea*, Britain's most serious poisonous weed to domestic livestock) after 2,4-D application (Irvine et al. 1977). Increased palatability was thought to be related to an increased water-soluble carbohydrate content. The authors reported that 2,4-D also may have increased the total unsaturated pyrrolizidine alkaloid content, thus increasing the plant's toxicity. Based on the results of this study, it was suggested that cattle be withheld from pastures for about 3 weeks after application of 2,4-D. Effects on grazing wildlife have not been reported.

Based on studies with honey bees, insects appear to be relatively tolerant to high levels of 2,4-D (USDA 1984). The LD_{50} of 2,4-D for honey bees ranged from 11.525 kg/bee for an unspecified route of exposure to 105 µg/bee administered orally (USDA 1984). Bees fed purified 2,4-D had decreased lifespans (approximately half the lifespan of bees exposed to lower doses) at 1,000 ppm; however, lifespans were not shortened in bees fed up to 1,000 ppm of the butoxyethanol ester, isooctyl ester, or the dimethylamine salt of 2,4-D (USDA 1984). A temporary decrease in reproductive rate was observed in bees fed 100 ppm or more of an unspecified 2,4-D formulation (presumed to be an acid), although no effects were observed at 10 ppm. The effect was reversible and abated when exposure was stopped (USDA 1984).

Table E6-5. Acute Oral Toxicity of 2,4-D to Mammals and Birds

Species	Form of 2,4-D	LD$_{50}$ (mg/kg)
Rat	Acid	375[a]
	Butyl ester	620[a]
Mouse	Acid	368[a]
	Butyl ester	380[a]
Guinea Pig	Acid	469[a]
	Butyl ester	848[a]
Rabbit	Acid	800[a]
	Butyl ester	424[a]
Dog	Acid	100[a]
Cat	Butyl ester	820[a]
Cattle	Butyl ester	100[a]
Mule Deer (8-11 months)	Acid	400-800[b]
Chicken	Acid	541[a]
	Butyl ester	2,000[a]
Mallard (4 months)	Acid	>2,000[b]
(3-5 months)	Sodium salt	>2,025[b]
Pheasant (3-4 months)	Acid	472[b]
Pigeon	Acid	668[a]
Japanese Quail (2 months)	Acid	668[b]
Chukar (4 months)	Acid	200-400[b]

[a]USDA 1984.
[b]Hudson et al. 1984.

Dalapon

Based on acute oral LD$_{50}$s ranging from 3,860 mg/kg in rabbits and guinea pigs to 7,570 mg/kg in rats, dalapon is of low toxicity to wildlife (EPA 1984d) (Table E6-6). Dalapon causes slight to moderate dermal and eye irritation in rabbits (EPA 1984d, USDA 1984). Cattle given oral capsules of 500 mg/kg for 10 days exhibited no adverse effects (Kenaga 1974, as cited in USDA 1984). Calves given oral doses of 1,000 mg/kg for 10 days experienced diarrhea, anorexia, weight loss, slowed pulse rate, and mucous discharge (Paynter et al. 1960, as cited in USDA 1984).

Calves showed no adverse effects when fed grass and oats treated with up to 30 lb/acre (USDA 1984).

Sheep given daily oral capsules of 100 mg/kg for 10 and 481 days showed 6 percent and 10 percent weight loss, respectively (Kenaga 1974, as cited in USDA 1984). However, in another study, sheep given daily oral doses of 100 mg/kg for 35 days showed no ill effects (Paynter et al. 1960, as cited in USDA 1984). Sheep fed oats treated with up to 20 lb/acre exhibited no adverse effects (USDA 1984).

Dalapon caused no teratogenic or reproductive effects in rats (EPA 1984d). Dalapon is rapidly excreted in the urine after ingestion,

Table E6-6. Acute Oral Toxicity of Dalapon to Mammals and Birds

Species	LD_{50} (mg/kg)
Rat	7,570
Mouse	4,600
Guinea pig	3,860
Rabbit	3,860
Chicken	5,660

Source: EPA 1984d.

and very few residues remain in tissues of animals (USDA 1984).

Dalapon is very slightly toxic to birds based on the acute oral LD_{50} of 5,660 mg/kg in chickens (EPA 1984d). The subacute dietary LC_{50} is greater than 5,000 ppm for Japanese quail, ring-necked pheasants, and mallard ducks (Hill et al. 1975, as cited in USDA 1984). The NOEL for chickens given oral doses of dalapon for 10 days is 100 mg/kg/day, based on decreased weight gain at the 250- and 500-mg/kg dose levels (Palmer and Radeleff 1969). Ten percent mortality was observed in adult pheasants given 5,000 ppm (250 mg/kg/day) in the diet for 10 days (USDA 1984). High mortality rates (up to 95 percent) were observed in pheasants, mallards, and bobwhites given 5,000 ppm in the diet for 24 to 169 days (USDA 1984).

Dalapon has been observed to cause depressed reproduction in mallards fed levels of less than 25 percent the lethal dose (USDA 1984). Female mallards and pheasants given 1,000 to 5,000 ppm in the diet laid fewer eggs, although female bobwhites fed the same amounts showed no change in the number of eggs laid (USDA 1984). No effects on hatching success or viability were observed in eggs of treated females. Chicken eggs injected with dalapon showed decreased hatching rates at concentrations of 300 ppm or more (Dunachie and Fletcher 1970, as cited in USDA 1984). The LC_{50} of mallard eggs immersed in an aqueous solution of dalapon was a concentration equivalent to a field application rate of greater than 375 lb/acre, which is more than 25 times greater than the field application rate (Hoffman and Albers 1984).

Dalapon is relatively nontoxic to honey bees. Honey bees momentarily dipped in a 20,000-ppm solution of dalapon showed no mortality after 24 hours (Kenaga 1974, as cited in USDA 1984). Bees fed 1,000 ppm dalapon by weight in sucrose showed an increased mortality, but bees fed 100 ppm dalapon showed no increased mortality compared to controls (Morton et al. 1972, as cited in USDA 1984).

Dicamba

Technical dicamba is slightly toxic to mammals based on oral LD_{50}s of 757 mg/kg in rats and 1,189 mg/kg in mice (USDA 1984). The oral LD_{50} for guinea pigs is 3,000 mg/kg and for rabbits 566 mg/kg (WSSA 1983). Technical dicamba caused mild dermal irritation and mild to moderate eye irritation in rabbits (EPA 1986b). The acute oral LD_{50} of the Banvel® formulation is 1,707 mg/kg in rats (USDA 1984). A study with Banvel showed that the chemical has a moderate potential for causing dermal sensitization in guinea pigs (EPA 1986b). Ten daily oral doses of 250 mg/kg of the Banvel D formulation caused no adverse effect in sheep (Palmer and Radeleff 1969). Two doses of 500 mg/kg of Banvel D caused death in sheep. However, one oral dose of 1,000 mg/kg caused no adverse effect in sheep (Palmer and Radeleff 1969).

Dicamba has not been observed to be teratogenic in rats and rabbits (EPA 1986b). In a three-generation reproduction study with rats, no reproductive effects occurred at the highest dose tested, 25 mg/kg/day (EPA 1986b). Dicamba is rapidly excreted in urine, primarily in its parent form, although some is excreted either as a conjugate with glucoronic acid or as 3,6-dichloro-2-hydroxybenzoic acid, and dicamba does not bioaccumulate in animal tissues (USDA 1984).

EPA (1983b) has characterized technical dicamba and formulated dicamba acid and its salts as slightly toxic to avian wildlife. The 8-day dietary LC_{50} of technical dicamba acid is greater than 10,000 ppm (1,750 mg/kg) in bobwhite quail and mallard ducks (EPA 1983c). An acute oral LD_{50} of 673 mg/kg was reported for technical dicamba in pheasants (USDA 1984). The acute oral LD_{50}s of the formulated products were all greater than 2,510 mg/kg in mallards, and 8-day dietary LC_{50}s were all greater than 4,640 ppm in mallards and bobwhite quail (EPA 1983c). Results of avian toxicity studies on formulated products of dicamba are summarized in Table E6-7.

No teratogenic effects were observed in chicken eggs injected with dicamba; however,

Table E6-7. Results of Avian Toxicity Studies With Formulated Dicamba

Formulation	Mallard	Bobwhite Quail
4 lb/gal dimethylamine salt (Banvel)	Oral LD_{50} >2,510 mg/kg Dietary LC_{50} >4,640 ppm	Dietary LC_{50} >4,640 ppm
1 lb/gal dimethylamine salt (Banvel CST)	Oral LD_{50} >2,510 mg/kg Dietary LC_{50} >5,620 ppm	Dietary LC_{50} >5,620 ppm
55% aluminum salt	Oral LD_{50} >2,510 mg/kg Dietary LC_{50} >5,620 ppm	Dietary LC_{50} >5,620 ppm
2 lb/gal sodium salt	Dietary LC_{50} >10,000 ppm	Dietary LC_{50} >10,000 ppm

Source: EPA 1983c.

hatching success was reduced at the highest dose tested of 400 ppm (USDA 1984). The LC_{50} of mallard eggs immersed in an aqueous solution of dicamba was greater than a concentration equivalent to a field application rate of 200 lb/acre (Hoffman and Albers 1984). However, eye malformations and stunted growth were observed at unspecified levels that were below the reported LC_{50} (Hoffman and Albers 1984).

Most invertebrate studies indicate that dicamba is moderately toxic to insects. The oral LD_{50} of dicamba for honey bees ranged from 3.6 µg/bee to greater than 10 µg/bee (USDA 1984). Contact studies with dicamba reported LD_{50}s of greater than 100 µg/bee and greater than 91 µg/bee (2.6 percent mortality was observed at 91 µg/bee) (USDA 1984). Such doses far exceed those encountered in the field because a field application of 1.12 kg/ha (1 lb/acre) would result in a contact dose equivalent to 1.25 µg/bee (Ghassemi et al. 1981). Ingestion of technical dicamba and the Banvel D4S formulation for up to 60 days had no effect on the mortality of honey bees at the highest dose tested of 1,000 ppm (Morton et al. 1972, as cited in USDA 1984). Cockroaches fed 1,000 ppm dicamba in food showed no developmental or reproductive effects (USDA 1984).

Based on current information, EPA (1983c) has concluded that dicamba is unlikely to directly affect wildlife species.

Diuron

Diuron is slightly toxic to mammals based on the acute oral LD_{50} of 3,400 mg/kg in rats (WSSA 1983). CNS depression was observed in mammals at high doses. Diuron was not

irritating to the eyes and skin of rabbits in primary irritation studies (EPA 1986c). Diuron did not cause teratogenic or reproductive effects in rats (EPA 1986c). According to Palmer and Radeleff (1969), application rates of up to 9.6 lb/acre would not be dangerous to sheep or cattle.

Diuron is very slightly toxic to birds based on the oral LD_{50} of greater than 2,000 mg/kg in mallard ducks (EPA 1983d). Signs of toxicity included incoordination and frequent falling (Hudson et al. 1984). The subacute dietary LC_{50} is greater than 5,000 ppm in mallards and ring-necked pheasants, and is 1,730 ppm in bobwhite quail (EPA 1983d). Diuron is toxic to chickens at relatively low doses (Palmer and Radeleff 1969). Chickens experienced decreased weight gain at the lowest dose tested of 10 mg/kg for 10 days. All chickens died after 9 doses of 250 mg/kg. Application rates of greater than 1 lb/acre were therefore considered hazardous to chickens.

Glyphosate

Glyphosate is generally recognized to be of low toxicity in the environment (USDA 1984). Acute oral LD_{50}s are 4,320 mg/kg for rats and 3,800 mg/kg for rabbits (EPA 1984e, USDA 1984). Based on these values, glyphosate can be considered slightly toxic.

Oral LD_{50} values for the Roundup® and Rodeo® formulations in rats are 5,400 mg/kg and greater than 5,000 mg/kg, respectively (Monsanto 1983, 1985). The oral LD_{50} of Roundup for goats is 4,860 mg/kg (Monsanto 1985). Glyphosate, Roundup, and Rodeo are reported to be practically nonirritating or slightly irritating to rabbits' eyes and skin (Monsanto 1983, 1985). Based on a 26-month

feeding study, a NOEL of greater than 31 mg/kg/day was established for rats (EPA 1986d). In a 1-year oral study with dogs, a NOEL of 500 mg/kg/day (HDT) was determined (EPA 1987b). Glyphosate has caused no reproductive or teratogenic effects in rats or rabbits (EPA 1984e).

Studies conducted on black-tailed deer in pens in the Pacific Northwest showed no gross adverse health effects caused by the use of glyphosate for vegetation management (Sullivan 1985). Glyphosate-treated browse and commercial chow were as acceptable by deer for consumption as untreated food. Likewise, glyphosate-induced weed and shrub control did not adversely affect deer use of treated habitat areas for at least the first year after treatment.

In a study to evaluate the direct effects of glyphosate on small mammals, no adverse effects on reproduction, growth, or survival were observed in populations of deer mice during the year following treatment (Sullivan 1985).

Glyphosate is slightly toxic to birds based on the acute oral LD_{50} of greater than 2,000 mg/kg in bobwhite quail (EPA 1986e). The 8-day dietary LC_{50} is more than 4,000 ppm for mallard ducks and bobwhite quail (EPA 1986e). Avian reproduction studies yielded no reproductive effects at dietary exposure levels of up to 1,000 ppm (EPA 1986e).

Residue and metabolism studies have indicated that glyphosate is incompletely absorbed across the gastrointestinal membranes and that in the vertebrates tested, there is minimal metabolism or retention by tissues and rapid elimination of residues (Monsanto 1982).

Glyphosate and Roundup are relatively nontoxic to insects based on the 48-hour acute oral and topical exposure toxicity of 100 μg/bee in honey bees (USDA 1984).

Hexazinone

Based on toxicity data for birds and mammals, hexazinone presents a low hazard to wildlife species (EPA 1982). The acute oral LD_{50} of technical hexazinone is 1,690 mg/kg in rats, 860 mg/kg in guinea pigs, and 2,258 mg/kg in bobwhite quail (EPA 1984f, EPA 1982). The acute oral LD_{50} of a 25-percent hexazinone solution is 6,887 mg/kg in rats (Du Pont 1984a). The 8-day dietary LC_{50}s of greater than 10,000 ppm for mallards and greater than 5,000 ppm for bobwhite quail indicate that

technical hexazinone is practically nontoxic to birds (EPA 1982). Formulated and unformulated hexazinone were irritating to the eyes but not to the skin of rabbits and guinea pigs (USDA 1984, EPA 1982). Hexazinone has not been observed to cause teratogenic or reproductive effects in rats or rabbits (EPA 1984f, USDA 1984). No appreciable bioaccumulation of hexazinone occurs in animal tissues (USDA 1984). Hexazinone is readily metabolized and is rapidly excreted in the urine and feces of animals (USDA 1984).

Hexazinone is relatively nontoxic to insects (Du Pont 1984a). The LD_{50} of a topical application of a 90-percent soluble powder of hexazinone is greater than 60 μg/bee for honey bees (Du Pont 1984a). A 60/mg/bee topical application rate resulted in 10 percent mortality in 48 hours (Schneider 1984 and Du Pont undated, both as cited in USDA 1984).

Imazapyr

Imazapyr is slightly toxic to mammals based on acute oral LD_{50}s ranging from greater than 2,000 mg/kg in mice to greater than 5,000 mg/kg in rats (Table E6-8) (EPA 1985e, American Cyanamid Company 1985). Technical imazapyr and the Arsenal[®] formulation are reported to be irritating to the eyes and mildly irritating to the skin of rabbits but are reported as nonsensitizing to guinea pigs (EPA 1985e, American Cyanamid Company 1985). No teratogenic effects were observed in rats or rabbits (American Cyanamid Company 1985). Imazapyr does not appear to accumulate in animal tissues (EPA 1985e).

EPA (1985b) characterizes imazapyr as practically nontoxic to avian species. Acute

Table E6-8. Acute Oral Toxicity of Imazapyr to Mammals and Birds

Species	LD_{50} (mg/kg)
Rat	>5,000[a]
Mouse	>2,000[a]
Rabbit	>4,800[a]
Bobwhite Quail	>2,150[b]
Mallard Duck	>2,150[b]

[a]American Cyanamid Company 1985.
[b]EPA 1985e.

oral LD_{50}s of technical imazapyr and the Arsenal formulation are greater than 2,150 mg/kg (HDT) in bobwhite quail and mallards (Table E6-8) (American Cyanamid Company 1985, EPA 1985b). Dietary LC_{50}s for formulated and unformulated imazapyr are greater than 5,000 ppm (HDT) for mallards and bobwhites (American Cyanamid Company 1985). No adverse effects were observed at any of these doses.

Imazapyr appears to be relatively nontoxic to insects. The LD_{50}s of technical imazapyr for honey bees is greater than 100 μg/bee (HDT) (American Cyanamid Company 1985). No effects were observed at this dose.

Mefluidide

Mefluidide is of low toxicity to mammals and birds based on acute oral LD_{50}s of 1,920 mg/kg in mice, greater than 4,000 in rats, and greater than 4,640 mg/kg in mallards (Worthing 1983, WSSA 1983). Mallard ducks and bobwhite quail, observed on the eighth day, had a 5-day dietary LC_{50} of greater than 10,000 mg/kg (Worthing 1983). Toxicity studies on other terrestrial wildlife species were not found.

Researchers have investigated mefluidide's effect on forage quality and on the quality of livestock feeding on treated forage (Wimer et al. 1986, Robb et al. 1982, Glenn et al. 1987). Effects on livestock reported in these studies were secondary effects related to mefluidide's effect on forage quality. No direct toxic effects from mefluidide were observed.

Mefluidide has been observed to affect population, development, or food choice of pest insects, such as the Mexican bean beetle (Agnello et al. 1986a), bean leaf beetle, corn earworm (Agnello et al. 1986b), and azalea lace bug (Coffelt and Schultz 1988). Population, feeding, or rate of development of these insects on treated plants decreased initially after application of mefluidide but later recovered or increased. These effects were hypothesized to be related to mefluidide-induced changes in plant nutritional quality rather than any direct toxicity to the insects.

In a 28-day feeding study on 12 lactating dairy cows, oral mefluidide treatments of 0, 6, 18, or 60 ppm of the cows' diet per day had no apparent effect on the general health, appearance, feed consumption, milk production, or quality of the carcasses at slaughter (Clark et al. 1981). With one exception, milk samples from cows fed at 6- and 8-ppm dose levels contained mefluidide

residues at levels less than 0.005 ppm. At the 60-ppm dose level, milk from test cows contained residues ranging from less than 0.005 to 0.015 ppm. Residues did not accumulate in milk, brain, muscle, liver, adipose tissue, or blood, and urinary elimination probably explains the mefluidide residues ($\leq$0.38 ppm) found in kidney samples.

Mefluidide is nontoxic to bees (Agrochemicals Handbook 1989).

Metsulfuron Methyl

Metsulfuron methyl is of low toxicity to birds and mammals based on acute oral LD_{50}s of greater than 5,000 mg/kg in rats and greater than 2,510 mg/kg in mallard ducks (Du Pont 1984b). The 8-day dietary LC_{50} for mallard ducks and bobwhite quail is greater than 5,620 mg/kg (Du Pont 1984b). Toxicity values for other terrestrial wildlife were not available.

Picloram

Picloram is slightly toxic to mammals based on acute oral LD_{50}s ranging from greater than 540 mg/kg in calves to 4,012 mg/kg in rats (Table E6-9) (Jackson 1966, EPA 1988). Technical picloram caused mild eye and skin irritation in rabbits (EPA 1984g). Picloram was not teratogenic in rats at the highest dose tested of 1,000 mg/kg (EPA 1984g). In a study by John-Greene et al. (1985), picloram was not teratogenic in rabbits at 400 mg/kg (HDT). The Tordon 101® formulation caused no ill effects in sheep at single doses of 1,900 mg/kg, but it caused death at levels of 2,200 mg/kg and above (Lynn 1965). Temporary weight loss was the only adverse effect seen in calves given Tordon 101 in single doses of 1,900 to 3,163 mg/kg (Lynn 1965). No toxic signs or adverse effects on growth were observed in sheep given 18 mg/kg/day of technical picloram in the diet for 33 days (Jackson 1966). Stimulated growth and improved feed efficiency were observed in swine given 22 mg/kg of feed for an unspecified time (McCollister and Leng 1969). Metabolic and residue studies in mammalian species indicate that picloram is rapidly eliminated unchanged in the urine following ingestion (USDA 1984). No metabolites have been detected (USDA 1984). In addition, picloram does not appear to accumulate to any significant extent in animal tissues (USDA 1984).

Picloram is slightly toxic to birds based on LD_{50}s that range from greater than 2,000 mg/kg in mallards and pheasants to approximately 6,000 mg/kg in chickens

(Table E6-5) (Lynn 1965, Hudson et al. 1984). Regurgitation occurred shortly after mallards were treated, and pheasants exhibited tremors and mild decline of muscle coordination after treatment (Hudson et al. 1984). Subacute dietary $LC_{50}s$ for bobwhite and Japanese quail, ring-necked pheasants, and mallard ducks were all greater than 5,000 ppm (NLM 1987a). The 8-day dietary LC_{50} of the Tordon 101 formulation is greater than 10,000 ppm for bobwhite quail and mallard ducks (EPA 1984h).

Japanese quail given 100 ppm in a 2-week dietary study showed no effects on feathering, reproduction, mortality, and weight (Kenaga 1969). In a similar test at 1,000 ppm, egg fertility and hatchability were reduced the first week but not the second (Kenaga 1969). A three-generation study with Japanese quail showed no effects on food consumption, reproduction, survival, and body weight when given 100, 500, or 1,000 ppm in the diet (Kenaga 1969). In a 1-year study in which Japanese quail were given 100 ppm to 10,000 ppm in their diet, no effects on reproduction, feeding, or body weights were observed. Mortality rates of treated quail were lower than those of controls (Kenaga 1969).

The LC_{50} of mallard eggs immersed in an aqueous emulsion of picloram was equivalent to a field application rate of 112 kg/ha (100 lb/acre), which is more than 10 times the recommended field application level (Hoffman and Albers 1984). Spray treatment of fertile chicken eggs or ring-necked pheasant eggs with a dose equivalent to 2.8 kg/ha (2.5 lb/acre) of Tordon 101 did not affect embryonic development or subsequent growth of hatched chicks (EPA 1984h).

Picloram is relatively nontoxic to insects based on an acute contact LD_{50} of greater than 14 µg/bee in honey bees (Kenaga 1979). Honey bees given 1,000 ppm picloram in a 60-percent sucrose syrup showed no toxic effects after 14 days and no increase in mortality compared to the control group after 60 days (USDA 1984).

Simazine

Simazine is slightly toxic to birds and mammals based on acute oral $LD_{50}s$ ranging from 2,010 mg/kg in gray-tailed voles to greater than 5,000 mg/kg in rats, mice, and rabbits (Table E6-10) (EPA 1983e, USDA 1984). Simazine is slightly irritating to the eyes but not to the skin of rabbits (USDA 1984). No teratogenic or reproductive effects have been observed in rats (EPA 1986f). Subchronic oral toxicity studies in cattle showed weight loss and toxic symptoms such as depression, muscular spasms, incoordination, and weakness when given doses ranging from 20 mg/kg/day for 6 days to 50 mg/kg/day for 10 days (Palmer and Radeleff 1969). Cattle given 7 doses of

Table E6-9. Acute Oral Toxicity of Picloram to Mammals and Birds

Species	LD_{50} (mg/kg)
Rat	4,012[a]
Mouse	2,000-4,000[a]
Rabbit	@2,000[a]
Guinea Pig	@3,000[a]
Sheep	>720[b]
Cattle	>750[d]
Calf	>540[b]
Chicken	@6,000[a]
Mallard Duck	>2,000[c]
Pheasant	>2,000[a]

[a]Hudson et al. 1984.   [d]USDA 1984.
[b]Jackson 1966.   [e]EPA 1988.
[c]Lynn 1965.

Table E6-10. Acute Oral Toxicity of Simazine in Birds and Mammals

Species	LD_{50} (mg/kg)
Rat	>5,000
Mouse	>5,000
Rabbit	>5,000
Prairie Vole	3,920 (males) 3,980 (females)
Gray-Tailed Vole	2,010 (males) 2,690 (females)
Mallard Duck	>4,640

Source: USDA 1984; EPA 1983e.

100 mg/kg/day became moribund; however, cattle given 3 doses of 250 g/kg/day showed weight loss but no other toxic signs. Sheep showed signs of intoxication and weight loss when given one dose of 250 mg/kg and 10 doses of 50 mg/kg/day (Palmer and Radeleff 1969). Sheep died when given 10 doses of 100, 250, or 400 mg/kg/day. Simazine did not bioaccumulate and was rapidly excreted in the urine and feces in rat metabolism studies (EPA 1986f).

The acute oral LD_{50} for Mallard ducks is greater than 4,640 (EPA 1983e). Dietary LC_{50}s of greater than 5,000 ppm in bobwhite quail, ring-necked pheasants, and mallards and greater than 3,720 ppm in Japanese quail indicate low toxicity to birds (EPA 1983e). An 11-week dietary study reported an LC_{50} of greater than 2,000 ppm for bobwhite quail. LC_{50}s of 11,000 ppm and greater than 32,000 ppm were reported for the 80-percent WP formulation in bobwhite quail and mallards, respectively. In a one-generation reproduction study with mallards, no reproductive impairment occurred at 20 ppm, the highest dose tested (EPA 1983e). Chicken eggs injected with simazine in an acetone solution exhibited decreased hatchability at 300 ppm, but not at 200 ppm or less (Dunachie and Fletcher 1970, as cited in USDA 1984). Based on the available information, EPA (1983e) concluded that simazine was practically nontoxic and will not affect reproduction in birds.

Sulfometuron Methyl

Sulfometuron methyl is very slightly toxic to birds and mammals based on acute oral LD_{50}s of greater than 5,000 mg/kg in rats and mallard ducks (EPA 1984i, Du Pont 1983). It is slightly irritating to rabbit eyes and skin but is nonsensitizing to guinea pigs (EPA 1984i). No teratogenic effects have been observed in rats and rabbits exposed to sulfometuron methyl (EPA 1984i); however, lower maternal body weights and decreased numbers of offspring were observed at 250 mg/kg/day in a reproduction study in rats (Du Pont 1986). The 8-day dietary LC_{50}s are greater than 5,620 ppm in bobwhite quail and greater than 5,000 ppm in mallards (Du Pont 1983). The LD_{50} was greater than 12.5 µg/bee when sulfometuron methyl was applied directly to bees (O'Neal 1987). No other studies have been reported on the toxicity of sulfometuron methyl to wildlife or insect species.

Tebuthiuron

Tebuthiuron is moderately to slightly toxic to mammals and birds based on acute oral LD_{50}s ranging from 286 mg/kg in rabbits to greater than 2,000 mg/kg in mallards and bobwhites (Table E6-11) (EPA 1986g, USDA 1986). Tebuthiuron is slightly irritating to the eyes but not to the skin of rabbits (EPA 1986g). It caused decreased body weight in weaning pups in a three-generation rat reproduction study at doses of approximately 20 mg/kg (LDT) (EPA 1986g). In other studies, however, no teratogenic effects were observed in rats at doses of approximately 90 mg/kg (HDT) or in rabbits at 25 mg/kg (HDT) (EPA 1986g). In subchronic oral toxicity studies, dogs experienced increased thyroid and spleen weight at 25 mg/kg (EPA 1986g). Decreased body weight was observed in cattle at 100 ppm in a 162-day study (EPA 1986g). Tebuthiuron was readily metabolized and eliminated in the urine of tested animals (USDA 1986).

In subacute oral toxicity studies, doses of up to 1,500 ppm resulted in no deaths in mallards and bobwhites (Meyerhoff 1981, as cited in USDA 1986). In a 30-day oral study, chickens exhibited depressed growth at 2,500 ppm (EPA 1986g). In 18-, 24-, and 27-week studies, no effects on growth, reproduction, or behavior were observed in bobwhite quail or mallard

Table E6-11. Acute Oral Toxicity of Tebuthiuron to Mammals and Birds

Species	LD_{50} (mg/kg)
Rat	644[a]
Mouse	528[b]
Rabbit	286[b]
Dog	>500[b]
Cat	>200[b]
Bobwhite Quail	>2,000[b]
Mallard Duck	>2,000[b]
Chicken	>500[b]

[a] EPA 1986g.
[b] USDA 1986.

ducks when fed up to 100 ppm in the diet (Elanco Products Company 1983, undated, as cited in USDA 1986).

Honey bees sprayed with 30,000 ppm tebuthiuron, which is equivalent to 5.56 kg/ha (5 lb/acre), did not differ in survival from bees sprayed with water. Bees sprayed with 120,000 ppm, equivalent to 22.4 kg/ha (20 lb/acre), had significantly higher mortality than controls (USDA 1986). Based on these results, tebuthiuron appears to be of relatively low toxicity to terrestrial invertebrates.

Triclopyr

Triclopyr is moderately toxic to mammals based on LD_{50} values that range from 310 mg/kg in guinea pigs to 729 mg/kg in male rats (EPA 1985b) (Table E6-12). Technical triclopyr is slightly irritating to the eyes and skin of rabbits (EPA 1985b). The Garlon 3A[®] and Garlon 4[®] formulations are slightly toxic, with oral LD_{50}s of 2,830 and

2,140 mg/kg in rats (males and females, respectively) (Dow Chemical Company undated). Garlon 3A may cause slight to moderate skin irritation and is moderately to severely irritating to eyes, and Garlon 4 may cause slight skin irritation but no eye irritation (Dow Chemical Company undated). Ponies exposed to four daily doses of 60 mg/kg of triclopyr exhibited no adverse effects; however, exposure to four daily doses of 300 mg/kg caused depression, recumbency, decreased gastrointestinal activity, and respiratory and muscular distress (Osweiler 1983).

No teratogenic effects have been observed in rats (EPA 1985b), but a rabbit study reported fetotoxic effects at the lowest dose of 10 mg/kg/day (EPA 1986h). Triclopyr is rapidly excreted, primarily as the parent compound, through the kidneys in animals (USDA 1984). Small quantities of two other compounds (the metabolite trichloropyridinol and a conjugated form of the parent, triclopyr acid) are also excreted (USDA 1984). Triclopyr does not

Table E6-12. Acute Toxicity of Triclopyr to Mammals and Birds

Species	Formulation	Test	Results
Rat	Technical	Oral LD_{50}	729 mg/kg (male)[a] 630 mg/kg (female)[a]
Mouse	Technical	Oral LD_{50}	471 mg/kg[b]
Rabbit	Technical	Oral LD_{50}	550 mg/kg[b]
Guinea Pig	Technical	Oral LD_{50}	310 mg/kg[b]
Rat	Garlon 3A	Oral LD_{50}	2,830 mg/kg (male)[a] 2,140 mg/kg (female)[a]
	Garlon 4	Oral LD_{50}	2,460 mg/kg (male)[a] 2,140 mg/kg (female)[a]
Mallard Duck	Technical	Oral LD_{50} Dietary LC_{50}	1,698 mg/kg[c] >5,640 ppm[a]
	Garlon 3A	Dietary LC_{50}	>10,000 ppm[a]
	Garlon 4	Oral LD_{50} Dietary LC_{50}	>4,640 mg/kg >10,000 ppm[a]
Japanese Quail	Technical	Dietary LC_{50}	3,278 ppm[c]
Bobwhite Quail	Technical	Dietary LC_{50}	2,935 ppm[c]
	Garlon 3A	Dietary LC_{50}	11,622 ppm[a]
	Garlon 4	Dietary LC_{50}	9,026 ppm[a]

[a]Dow Chemical Company undated.
[b]EPA 1985b.
[c]Kenaga 1979.
[d]Dow Chemical Company 1987.

bioaccumulate in animal tissues in any significant amount (Dow Chemical Company 1987).

Based on acute oral and dietary studies, triclopyr, Garlon 3A®, and Garlon 4® are slightly toxic to birds (Table E6-12). The acute oral LD_{50} of technical triclopyr is 1,698 mg/kg for mallard ducks, and the dietary LC_{50} ranges from 2,935 to greater than 5,000 ppm (Dow Chemical Company undated, Kenaga 1979). The dietary LC_{50}s of Garlon 3A and Garlon 4 are all greater than 9,000 ppm (Dow Chemical Company undated). A one-generation reproduction study showed no reproductive effects, symptoms of toxicity, or abnormal behavior when mallards were given up to 500 ppm in their diet for a 20-week period, including 10 weeks before egg laying and 10 weeks during egg laying (Dow Chemical Company 1987). A similar study reported no reproductive or toxic effects in bobwhite quail exposed to dietary levels of up to 800 ppm for a 20-week period, including 11 weeks before egg laying and 8 weeks during egg laying (Dow Chemical Company 1987).

The acute contact LD_{50} of triclopyr in honey bees is greater than 60 µg/bee, indicating that it is relatively nontoxic to insects (Kenaga 1969). The contact LD_{50} for honey bees is greater than 100 µg/bee based on a 1985 study (Dow Chemical Company 1987).

Light Fuel Oil

Kerosene and diesel oil are very slightly toxic to mammals based on the acute oral LD_{50}s of greater than 28,000 mg/kg and 7,380 mg/kg, respectively, in rats (NLM 1987b, Beck et al. 1982). Toxic effects include loss of muscle coordination, nausea, languor, drowsiness, rapid heart beat, and shallow respiration (ITII 1976). Diesel oil is extremely irritating to the skin of rabbits but nonirritating to the eyes (Beck et al. 1982). Kerosene is mildly irritating to the skin and eyes of rabbits and nonsensitizing in guinea pigs (Beck et al. 1982). Dermal exposure to 6,560 mg/kg of diesel oil for 3 weeks caused a 67-percent mortality rate in rabbits (API 1982). Dermal exposure to kerosene for 28 days caused skin and liver lesions in rabbits at the highest dose tested of 2,000 mg/kg but not at the next highest dose of 1,000 mg/kg (API 1983). Other adverse effects to the skin of the treated animals were observed at all three doses tested (200, 1,000, and 2,000 mg/kg), including cracking, scab formation, necrosis, and ulcerations (API 1983). No teratogenic effects were observed in rats when exposed to kerosene and diesel vapors during gestation

(Mecler and Beliles 1979, Beliles and Mecler 1982).

Diesel oil is very slightly toxic to birds when ingested, based on the acute oral LD_{50} of greater than 16,400 mg/kg (greater than 20 ml/kg) in mallards (Hudson et al. 1984). The toxic effects included weakness, diarrhea, and regurgitation. However, diesel oil appears to cause adverse reproductive effects in birds. Traces of oil in a mallard's diet sharply reduce egg production (Biderman and Dury 1980, as cited in DOE 1983). Application of only 1 microliter (µl) of No. 2 fuel oil on mallard eggs significantly reduced survival and hatchability (Szaro et al. 1978). In the same study, application of 5 µl reduced hatching success to 18 percent, and 20 µl killed all embryos. Similar toxicity was noted in pheasant eggs sprayed with diesel oil to runoff, which failed to hatch (Kopischke 1972). Death appears to be related to the aromatic portion of the oil rather than the aliphatic portion (Szaro et al. 1978, Hoffman and Albers 1984). In addition, oil carriers increase the toxicity of pesticides to eggs, apparently by increasing penetration through the shell and membrane (Hoffman and Albers 1984).

No acute oral LD_{50} of kerosene to mallards was found, however, the LD_{50} level is not expected to be below the 16,400-mg/kg level found for diesel oil in mallards. Kerosene was not lethal when applied to mallard eggs at doses of 1 to 50 µl/egg (Hoffman and Albers 1984). The low toxicity observed in this study was believed to be related to the lower aromatic hydrocarbon content of kerosene (Hoffman and Albers 1984).

Diesel oil is highly toxic to insects based on high mortality of honey bees during the first 24 hours after spray treatment (Moffet et al. 1972). No information was available on the toxicity of kerosene to honey bees. Kerosene and diesel oil, when used as solvents or adjuvants, also have been observed to increase the toxicity of insecticides (Lagier et al. 1974, Tsuda and Okuno 1985).

Aquatic Species Hazard Analysis

The toxicity to aquatic species of the herbicides and additives proposed for BLM use for vegetation management is summarized in this section. Information is presented on the acute and chronic toxicities of the herbicides to fish, aquatic invertebrates, and amphibians.

The relative acute toxicities of the herbicides are classified according to a scheme by EPA

(1985c) where LC_{50} values are described as follows: <0.1 ppm (1 ppm = 1 mg/l), very highly toxic; 0.1 ppm to 1 ppm, highly toxic; >1 ppm to ≤10 ppm, moderately toxic; >10 ppm to ≤100 ppm, slightly toxic; and >100 ppm, practically nontoxic.

The information presented in this section is used in the Aquatic Risk Analysis section (in Section 8) as a basis for selecting toxicity values for organisms representative of the aquatic environments in the 13 Western States. In some cases, a number of toxicity tests have been conducted under various water quality conditions with a particular herbicide and a given species that have resulted in a range of LC_{50} values (for example, technical grade picloram and rainbow trout in Mayer and Ellersieck 1986). In these cases, the lowest reported value from the range has been included in the table in the hazard analysis.

The terms listed below pertain to aquatic toxicology and are used frequently in this section:

LC_{50}—the concentration of a toxicant in water that is lethal to 50 percent of a population of test organisms within a specific period of time (usually reported for 96 hours).

EC_{50}—the concentration of a toxicant in water that has a specific effect on 50 percent of the test organisms. It is often used with animals where determining death is difficult, such as with Daphnia sp. In this case, immobilization of an animal is the measured endpoint.

MATC—maximum acceptable toxicant concentration, which is the hypothetical toxic threshold concentration of a toxicant in water bounded by the highest tested concentration that has no significant adverse effect and the lowest concentration having a significant effect.

Static test—toxicity tests (generally only acute tests) in which the solution in the test chamber is still (not flowing); the solution may be renewed during the course of the test.

Flow-through test—toxicity test (acute, subchronic, or chronic) in which the solution in the test chamber is flowing continuously or intermittently. Flow-through tests generally result in somewhat lower LC_{50}s than static tests conducted under the same conditions.

Amitrole

The toxicity of amitrole to aquatic organisms is summarized in Table E6-13. Amitrole is only slightly toxic to fish and crayfish, with LC_{50}s generally greater than 100 ppm. Microcrustaceans, such as cladocerans (water fleas) and copepods, are much more sensitive; the lowest reported 96-hour LC_{50} is 1.43 ppm for Daphnia magna.

A formulation of 50 percent amitrole (Weedazol TL Plus®) was of low toxicity to frog tadpoles; the 96-hour LC_{50} was 3,000 ppm (Johnson 1976, as cited in USDA 1984).

Atrazine

Atrazine is moderately to highly toxic to fish and aquatic invertebrates (Table E6-14). Early life-stage studies with fish and amphibians have indicated that atrazine is highly acutely toxic and teratogenic. Atrazine also affects reproduction in a number of invertebrates at concentrations of 1 ppm or less.

Bromacil

Bromacil is slightly toxic to aquatic organisms. All of the LC_{50} values in Table E6-15 are 40 ppm or greater. The most sensitive organisms tested were water fleas and crayfish. The 48-hour LC_{50} for tadpoles is 230 ppm.

Chlorsulfuron

Chlorsulfuron is practically nontoxic to fish based on 96-hour LC_{50}s of greater than 250 mg/L for bluegill and rainbow trout (Du Pont 1982) (Table E6-16). Toxicity values for amphibians and aquatic invertebrates were not available.

Clopyralid

Clopyralid appears to have a low order of toxicity to fish and aquatic invertebrates. Clopyralid is practically nontoxic to trout and bluegill with 96-hour LC_{50}s of 103.5 mg/L and 125.4 mg/L, respectively (WSSA 1983) (Table E6-16). Daphnia showed no mortality in a 1-mg/L solution of clopyralid (WSSA 1983). Toxicity values for amphibians were not available.

2,4-D

The aquatic toxicity of the butoxyethanol ester of 2,4-D ranges from moderately to highly toxic (Table E6-17). Acute LC_{50} values range from about 0.5 ppm to 10 ppm for most species. Amphipods and snails are among the most sensitive groups. Esters are typically 100 times more toxic than their corresponding

Table E6-13. Toxicity of Amitrole to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Salmon	3,250	48-hr LC_{50}	Bohmont 1967[a]
Bluegill	1,200	24-hr LC_{50}	Lorz et al. 1979[b]
Fathead minnow	>100	96-hr LC_{50}	Mayer and Ellersieck 1986
Channel catfish	>160	96-hr LC_{50}	Mayer and Ellersieck 1986
Mosquito fish (Amitrol-T®)	2,100	96-hr LC_{50}	Johnson 1978[b]
Emerald shiner	455 (Cytrol Amitrol-T®)	24-hr LC_{50}	USDA 1984
Crayfish *Orconectes*	>100 (Amitrol-T®)	48-hr LC_{50}	Sanders 1970[b]
Brine shrimp	5	Reduced zygote production at 31-41 days.	Grosch 1980[b]
Water flea *Daphnia magna*	1.43 23	96-hr LC_{50} 26-hr EC_{50} (based on immobilization)	USDA 1984
Copepod *Cyclops vernalis* -nauplii larvae	22.1 (18.5-27.7)	96-hr LC_{50}	USDA 1984
Aquatic sowbug *Asellus* sp.	>100 (Cytrol Amitrol-T®)	96-hr LC_{50}	Johnson and Finley 1980
Tadpole *Adelotus brevis*	3,000 (Weedazol TL® plus)	96-hr LC_{50}	Johnson 1976[b]

[a] As cited in Pimentel 1971.
[b] As cited in USDA 1984.

Table E6-14. Toxicity of Atrazine to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	24 (18-32) (atrazine 4 L)	96-hr LC_{50} (95% C.L.)	Mayer and Ellersieck 1986
	4.5 (3.5-5.7) (technical)	96-hr LC_{50}	Bathe et al. 1976[a]
- fry	0.87 (0.63-1.15)	96-hr LC_{50} Teratogenic effects in 3, 6, and 62% at 0.05, 0.54, and 5.02 ppm, respectively. Exposure from spawning to 96 hours post hatching.	Birge et al. 1979[a] and 1983
Brook trout	6.3 (4.1-9.7)	96-hr LC_{50}	Macek et al. 1976[e]
- fry	0.24	Reduced survival and growth.	Macek et al. 1976[e]
- adults	0.72	No effect on survival, egg production, or egg hatching.	Macek et al. 1976[e]
Bluegill	42 (36-39) (atrazine 4 L)	96-hr LC_{50} (95% C.L.)	Mayer and Ellersieck 1986
	0.5	Fish became lethargic, fed poorly, and had partial loss of equilibrium after 28 days.	Macek et al. 1976[e]
	6 (approx.) (wettable power)	96-hr LC_{50}	USDA 1984
- eggs & fry	0.095	No effects on hatching, survival, or growth after 18 months.	Macek et al. 1976[e]
Fathead minnow	15 (11-20)	96-hr LC_{50}	Macek et al. 1976[e]
- fry	0.52	25% mortality at 96 hours.	
	0.213	No effect on survival, growth, or spawning after 43 weeks.	

Table E6-14. Toxicity of Atrazine to Aquatic Organisms (continued)

Species	Concentration (ppm)	Effect	Source
Catfish	7.6 (atrazine)	96-hr LC_{50}	USDA 1984
	35 (80% wettable powder)	96-hr LC_{50}	USDA 1984
	0.22 (0.15-0.32)	96-hr LC_{50} Teratogenic effects in 4, 13, 69, and 100% at 0.06, 0.43, 4.83, and 46.7 ppm respectively. Exposure from spawning to 96 hours post hatching.	Birge et al. 1979 and 1983[a]
Brown shrimp	1.0	30% mortality or immobility at 48 hours.	Butler 1965[a]
Water flea	3.6	48-hr LC_{50}	USDA 1984
Daphnia magna	1.15	Decreased fecundity, no effect on survival.	Macek et al. 1976[a]
D. pulex	1.0	Decreased fecundity. Decreased survival at 20 ppm, not at 10. Synergistic effects with ethanol.	Schober and Lampert 1976 and 1977[a]
Scud Gammarus fasciatus	5.7 (3.6-8.0)	48-hr LC_{50}	Macek et al. 1976[a]
	0.14	Reproductive effects and reduced survival of offspring at exposures of 30 days to 17 weeks.	
Clam and snail	0.5	87.5% mortality in clams; snails increased by approximately 400%.	USDA 1984

Table E6-14. Toxicity of Atrazine to Aquatic Organisms (continued)

Species	Concentration (ppm)	Effect	Source
Midge *Chironomus tentans*	0.72 (0.36-1.44)	48-hr LC_{50}	Macek et al. 1976[a]
- larvae	0.11	No adverse effect after 2 generations; reduced hatching success, increased larval mortality, and retarded development at 0.23 ppm.	
Bullfrog - tadpoles	0.41	LC_{50} from spawning to 96 hours post hatching; Teratogenic effects of 3, 7, 22, 47, and 100% at 0.4, 6.33, 14.8 26.4, and 45.8 ppm, respectively.	Birge et al. 1980 and 1983[a]
Leopard frog - tadpoles	0.31	Significant decrease in growth from 27-45 days. Significant increase in mortality at 0.31 to 12 ppm from 27 to 54 days of exposure.	Hine et al. 1981[a]

[a]As cited in USDA 1984.

Table E6-15. Toxicity of Bromacil to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	75	48-hr LC_{50}	Sherman and Kaplan 1975
Bluegill	71	48-hr LC_{50}	Sherman and Kaplan 1975
Carp	164	48-hr LC_{50}	Sherman and Kaplan 1975
Crayfish	40	72-hr LC_{50}	EPA 1975
Water flea	40	3-hr LC_{50}	EPA 1975
Tadpole	230	48-hr LC_{50}	EPA 1975

Table E6-16. Toxicity of Chlorsulfuron, Clopyralid, Mefluidide, and Metsulfuron Methyl to Aquatic Organisms

Chemical	Species	Test	Result	Source
Chlorsulfuron	Rainbow trout	96-hr LC_{50}	>250 mg/l	Du Pont 1982
	Bluegill	96-hr LC_{50}	>250 mg/l	Du Pont 1982
Clopyralid	Trout	96-hr LC_{50}	103.5 mg/l	WSSA 1983
	Bluegill	96-hr LC_{50}	125.4 mg/l	WSSA 1983
	Daphnia magna	1 ppm solution	no mortality	WSSA 1983
Mefluidide	Rainbow trout	96-hr LC_{50}	>100 mg/l	Worthing 1983
	Bluegill	96-hr LC_{50}	>100 mg/l	Worthing 1983
Metsulfuron Methyl	Rainbow trout	96-hr LC_{50}	>150 mg/l	Du Pont 1984b
	Bluegill	96-hr LC_{50}	>150 mg/l	Du Pont 1984b
	Daphnia magna	48-hr LC_{50}	>150 mg/l	Du Pont 1984b

Table E6-17. Toxicity of 2,4-D Butoxyethanol Ester to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout			
- fingerlings	1.42 to 1.55	96-hr LC_{50}	Halter 1980
- yearlings	9.0	96-hr LC_{50}	Dodson and Mayfield 1979[a]
Bluegill	1.2	96-hr LC_{50}	Johnson and Finley 1980
Fathead minnow	3.3	96-hr LC_{50}	Johnson and Finley 1980
Black bullhead	7.1 to 7.7	96-hr LC_{50}	Halter 1980
Crayfish *Orconectes nais*	>100	48-hr LC_{50}	Sanders 1970[a]
Glass shrimp *Palaemonetes kadiakensis*	1.4	48-hr LC_{50}	Sanders 1970[a]
Shrimp	1.0	48-hrs, no effect	Ghassemi et al. 1981
Water flea *Daphnia pulex*	3.0	8 days, no effect	Sigmon 1979[a]
D. magna	5.6	48-hr LC_{50}	Sanders 1970[a]
Copepod *Nitocra spinipes*	3.1	96-hr LC_{50}	Linden et al. 1979[a]
Scuds *Gammarus lacustris*	0.44	96-hr LC_{50}	Sanders 1969
Gammarus fasciatus	5.9	96-hr LC_{50}	Sanders 1970[a]
Sowbug *Asellus brevicaudis*	2.6	96-hr LC_{50}	Johnson and Finley 1980
Seed shrimp *Cypridopsis vidua*	2.2	48-hr EC_{50}	Johnson and Finley 1980
	1.8	48-hr LC_{50}	Sanders 1970[a]
Stonefly *Pteronarcys californica*			
- adult	>1,000	96-hr LC_{50}	FWPCA 1968 in Pimental 1971
- nymphs	1.6	96-hr LC_{50}	Sanders and Cope 1968
Eastern oyster	3.75	96-hr EC_{50} decrease in shell growth	Butler 1965[a]
Snail *Lymnea* sp.	0.32	42% mortality at 6 weeks	Halter 1980

[a]As cited in USDA 1984.

acids and most amine formulations, but in most cases they rapidly hydrolyze to corresponding acids (Ghassemi et al. 1981). Bioaccumulation of 2,4-D is low, and it generally is rapidly excreted in the urine unchanged or as a conjugate (USDA 1984). 2,4-D amine is practically nontoxic to amphibians (Johnson 1976).

A 48-hour LC_{50} of 1.1 ppm (isooctylester) has been reported for adult bluegill (Pimentel 1971). This value compares closely with the 96-hour LC_{50} of 1.2 ppm for the butoxyethanol ester of 2,4-D for the same fish species (Table E6-18). Because of the close chemical similarities of the two herbicides, it is expected but not proven that their aquatic toxicities would be similar.

Dalapon

Dalapon is slightly toxic to fish based on the results of studies on eggs, fry, fingerlings, and adult fish (Table E6-18). Its toxicity to invertebrates is quite variable; some organisms, such as *Daphnia magna*, brown shrimp, and stonefly nymphs, are sensitive, whereas dragonflies, snails, and copepods are fairly tolerant. Tadpoles are very tolerant of diuron (96-hour LC_{50} = 4,200 ppm).

Dicamba

Dicamba is only slightly toxic to most aquatic organisms (Table E6-19). The salts and free acid of dicamba are considered toxicologically equivalent because the salt hydrolyzes to the free acid in an aqueous environment (EPA 1983b). Short-term LC_{50} values are greater than 10 ppm for fish, amphibia, and most invertebrates. The amphipod *Gammarus lacustris*, which has a 96-hour LC_{50} of 3.9 ppm, is more sensitive to dicamba than any other aquatic animal tested (Sanders 1969). A 48-hour EC_{50} of 11 ppm was determined for *Daphnia pulex* (Sanders and Cope 1966, as cited in USDA 1984). *Daphnia magna*, with a 48-hour EC_{50} of greater than 100 ppm (Johnson and Finley 1980) does not appear to be as sensitive as *D. pulex*. No long-term aquatic toxicity studies have been reported.

Yolk-sac fry, fingerlings, and eggs of salmonids are not acutely sensitive to fosamine (USDA 1984). The 96-hour EC_{50} based on avoidance behavior and white blood cell counts in coho salmon, also are greater than 100 ppm (USDA 1984). No toxicity studies with amphibians have been reported, and no long-term studies have been reported with aquatic organisms.

Diuron

Diuron is moderately toxic to fish (Table E6-20). The 96-hour LC_{50}s for technical diuron range from 1.4 ppm for cutthroat trout to 8.2 ppm for bluegill. Aquatic invertebrates are more sensitive to diuron than fish; the lowest LC_{50} is 0.16 ppm for the scud (*Gammarus fasciatus*.) Toxicity values for amphibians have not been reported.

Glyphosate

Roundup and Rodeo are the two formulations of glyphosate that BLM may use for vegetation management. Because of its surfactant content, Roundup is much more toxic to aquatic organisms than the other two formulations, which do not contain surfactants. Therefore, it is important to treat separately the risk of different formulations.

Roundup

The toxicity of the Roundup formulation (41 percent isopropylamine (IPA) salt of glyphosate, 15 percent surfactant, and 44 percent water) to aquatic organisms is summarized in Table E6-21. Roundup is moderately to slightly toxic; most 96-hour LC_{50} values range from 2 to 18 ppm. The acute toxicity of Roundup is greater at pH 7.5 than pH 6.5, and toxicity also increases with increasing temperature (Folmar et al. 1979). Rainbow trout did not exhibit avoidance behavior at concentrations up to 10 ppm, whereas mayfly nymphs showed avoidance behavior at this level (Folmar et al. 1979).

Rainbow trout were exposed for 12 hours to 0.02, 0.2, and 2.0 ppm of formulated Roundup (Folmar et al. 1979). No effects were observed on fecundity or maturation of gonads after being held in freshwater for 30 days. Midge larvae also were exposed to 0.02, 0.2, and 2.0 ppm of Roundup. Significant increases in stream drift of the larvae were observed at the highest concentration.

Rodeo

The Rodeo formulation (53.5 percent isopropylamine salt of the active ingredient N-phosphonomethyl glycine and 46.5 percent water) of glyphosate is practically nontoxic to aquatic organisms (Table E6-21). The 96-hour LC_{50}s for fish are all greater than 1,000 ppm, and the 48-hour LC_{50} for *Daphnia magna* is 930 ppm (Monsanto 1983).

Table E6-18. Toxicity of Dalapon to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	340 (sodium salt, powder 74% a.i.)	96-hr LC_{50}	Folmar 1976[a]
	1.0	avoidance behavior	
Bluegill	105 (technical)	96-hr LD_{50}	Mayer and Ellersieck 1986
	440 (Radapon®, sodium salt)	96-hr LC_{50}	USDA 1984
- fingerlings	>1,000 (granular and wettable powder)	48-hr LC_{50}	USDA 1984
Green sunfish			
- fry	>50	8-day LC_{50}	Hiltibran 1967[a]
- eggs	>50	3-day LC_{50}	
Fathead minnow	290-390 (Radapon®, sodium salt)	96-hr LC_{50} (toxicity greater in soft water than hard water)	USDA 1984
Channel catfish			
- fingerlings	10	Less than 10% mortality at 48 hours.	McCorkle et al. 1977[a]
Smallmouth bass			
- fry	>50	8-day LC_{50}	Hiltibran 1967[a]
Mosquito fish	>19,100	96-hr LC_{50}	Johnson 1978[a]
Grass carp	>30,000 (Dalapon 85% a.i.)	96-hr LC_{50} (No mortality observed)	Tooby et al. 1980
Emerald shiner	>3,000	72-hr LC_{50} (no toxic effect)	Springer 1957[b]
Lake chubsucker			
- fry	>50	8-day LC_{50}	Hiltibran 1967[a]
Brown shrimp	1.0 (sodium salt Radapon®)	48-hr EC_{50} 40% mortality or immobilization.	Butler 1965[a]

Table E6-18. Toxicity of Dalapon to Aquatic Organisms (continued)

Species	Concentration (ppm)	Effect	Source
Water fleas *Daphnia magna*	6	48-hr LC_{50}	FWPCA 1968[b]
Daphnia pulex	11 (8.2 - 14.7)	48-hr LC_{50}	Mayer and Ellersieck 1986
Simocephalus serrulatus	16 (11.4 - 22.4)	48-hr EC_{50}	Mayer and Ellersieck 1986
Copepods *Cyclops* sp.	400 (sodium salt)	100% mortality at 24 hrs; no mortality at 200 ppm.	Kenaga 1974[a]
Nitocra spinipes	4,800 (sodium salt, Nacam)	96-hr LC_{50}	Linden et al. 1979
Fairy shrimp *Eubranchipus* sp.	400 (sodium salt)	100% mortality at 24 hrs; no mortality at 200 ppm.	Kenaga 1974[a]
Stonefly *Pteronarcys* sp. - nymphs	1.0 (sodium salt)	24-hr LC_{50}	Cope 1965[b]
Pteronarcys californica	>1,000	96-hr LC_{50}	Mayer and Ellersieck 1986
Dragonfly *Aeschna* sp.	>1,600 (sodium salt a.e.)	No mortalities at 48 hours.	Kenaga 1974[a]
Snails *Planorbis* sp.	400 (sodium salt a.e.)	50% mortality; no mortality at 200 ppm.	Kenaga 1974[a]
Physa sp.	400 (sodium salt a.e.)	50% mortality; no mortality at 200 ppm.	Kenaga 1974[a]
Tadpoles *Adelotus brevis*	4,200 (Dowpon®)	96-hr LC_{50}	Johnson 1976[a]

[a]As cited in USDA 1984.
[b]As cited in Pimentel 1971.

Table E6-19. Toxicity of Dicamba (88% technical) to Aquatic Organisms

Species	96-hr LC$_{50}$ (ppm)	Source
Rainbow trout	28	Johnson and Finley 1980
- fingerlings, 0.8g	135	Velsicol Chem. Corp.[c]
Cutthroat trout	>50	Woodward 1982[d]
Coho salmon		Lorz et al. 1979[d]
- juveniles	120[a]	
Bluegill		
- fingerlings, 0.9g	>50	Johnson and Finley 1980
	135	Velsicol Chem. Corp.[c]
Grass shrimp Palaemonetes kadiakensis	>56	Johnson and Finley 1980
Water flea Daphnia sp.		Sanders and Cope 1966[d]
	11[b]	
Daphnia magna	>100[b]	Mayer and Ellersieck 1986
- 1st instar		
Scud Gammarus fasciatus	>100	Johnson and Finley 1980
Sowbug Asellus brevicaudus	>100	Johnson and Finley 1980
Frog, tadpole		
-(1-2 wks old) Adelotus brevis	185	Johnson 1976[d]
-(1-2 wks old) Limnodynastes peroni	106	Johnson 1976[d]

[a]48-hr LC$_{50}$.
[b]48-hr EC$_{50}$.
[c]As cited in Ghassemi et al. 1981.
[d]As cited in USDA 1984.

Table E6-20. Toxicity of Diuron to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	4.9 (4.1 - 5.9)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
	16 (11 - 23)[b]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
Lake trout	2.7 (2.4 - 3.0)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
Cutthroat trout	1.4 (1.1 - 1.9)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
Bluegill	8.2 (7.4 - 9.1)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
Water fleas Daphnia pulex	1.4 (1.0 - 1.9)[a]	48-hr EC_{50} (95% C.L.)	Johnson and Finley 1980
Simocephalus serrulatos	2.0 (1.4 - 2.8)[a]	48-hr EC_{50} (95% C.L.)	Johnson and Finley 1980
Scud Gammarus fasciatus	0.16 (0.13 - 0.19)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
Sowbug Asellus brevicaudus	15.5 (7.2 - 33.4)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980
Stonefly Pteronarcys californica	1.2 (0.9 - 1.7)[a]	96-hr LC_{50} (95% C.L.)	Johnson and Finley 1980

[a]Technical material, 95%.
[b]Wettable powder, 80%.

Table E6-21. Acute Toxicity of Glyphosate to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Roundup®:			
Rainbow trout			
-fingerlings, 1g	1.3	96-hr LC_{50}	Folmar et al. 1979
-fingerlings, 2g	7.8 to 14.0	96-hr LC_{50}	
Bluegill	1.8 to 4.2	96-hr LC_{50}	Folmar et al. 1979
Fathead minnow	2.3	96-hr LC_{50}	Folmar et al. 1979
Channel catfish			
-fingerlings	13.0	96-hr LC_{50}	Folmar et al. 1979
-swim-up fry	3.3	96-hr LC_{50}	
-adult	16.0	96-hr LC_{50}	Monsanto 1982
Grass carp	15.0	96-hr LC_{50}	Tooby et al. 1980
Carp	3.9	96-hr TL_{50}	Monsanto 1982
Crayfish	>1,000	96-hr LC_{50}	Monsanto 1982
Water flea Daphnia magna	3.0	48-hr LC_{50}	Folmar et al. 1979
Copepod *Nitocra spinipes*	22.0	96-hr LC_{50}	Linden et al. 1979
Scud *Gammarus pseudolimnaeus*	43.0	96-hr LC_{50}	Folmar et al. 1979
Midge larvae	18.0	48-hr EC_{50}	Folmar et al. 1979
Rodeo®:			
Trout	>1,000	96-hr LC_{50}	Monsanto 1983
Bluegill	>1,000	96-hr LC_{50}	Monsanto 1983
Carp	>10,000	96-hr LC_{50}	Monsanto 1983
Water flea *Daphnia magna*	930	48-hr LC_{50}	Monsanto 1982

Table E6-21. Acute Toxicity of Glyphosate to Aquatic Organisms (continued)

Species	Concentration (ppm)	Effect	Source
Glyphosate[b]:			
Rainbow trout	140 (120-170)	96-hr LC_{50}	Folmar et al. 1977[a]
	38	96-hr LC_{50}	USDA 1981[c]
Bluegill	140 (110-160)	96-hr LC_{50} (static test)	Folmar et al. 1977[a]
	24	96-hr LC_{90} (flow through test)	USDA 1981[c]
Fathead minnow	97 (79-120)	96-hr LC_{50}	Folmar et al. 1977[a]
Channel catfish	130 (110-160)	96-hr LC_{50}	Folmar et al. 1977[a]
Carp	115	96-hr LC_{50}	USDA 1981[c]
Water flea *Daphnia sp.*	780	40-hr LC_{50}	Monsanto 1982[c]
Midge *Chironomus plumosus*	55	48-hr EC_{50}	Folmar et al. 1979

[a]As cited in Ghassemi et al. 1981.
[b]Technical glyphosate (95% or more of active ingredient) is assumed to be the formulation used.
[c]As cited in USDA 1984.

Technical Glyphosate

Technical glyphosate is slightly to practically nontoxic to fish and invertebrates (Table E6-21). Studies with channel catfish, bluegill, rainbow trout, and largemouth bass indicate that glyphosate does not bioaccumulate in fish to any significant degree (Monsanto undated). The toxicity of glyphosate or glyphosate-formulations to amphibians has not been reported in the literature.

An MATC of greater than 25.7 ppm has been reported in a long-term study with fathead minnows (Monsanto undated). A 21-day study with *Daphnia magna* determined a NOEL of 50 ppm based on decreased reproduction (Monsanto undated).

Hexazinone

The aquatic toxicity of hexazinone is summarized in Table E6-22. Hexazinone is practically nontoxic to fish; all 96-hour $LC_{50}s$ are greater than 100 ppm. EPA (1982, as cited in USDA 1984) has described technical hexazinone as "practically nontoxic" to fish. It is slightly toxic to aquatic invertebrates (Table E6-13). A 21-day NOEL of 10 ppm (technical) has been determined for *Daphnia* sp. (Mayack et al. 1982 and EPA 1982, both as cited in USDA 1984). No toxicity studies have been reported for amphibians. No chronic studies with aquatic organisms have been reported.

Imazapyr

Technical imazapyr, the isopropylamine salt of imazapyr, and the Arsenal 2.0 AS formulation are practically nontoxic to rainbow trout, bluegill, and channel catfish (Table E6-23). The water flea, the only aquatic invertebrate that has been tested, was not sensitive to Arsenal (American Cyanamid Company 1985). No studies have been reported with amphibians. Chronic or reproductive studies have not been reported in the literature.

Mefluidide

Mefluidide is practically nontoxic to fish based on 96-hour $LC_{50}s$ of 1,600 mg/L for bluegill and greater than 1,200 mg/L for trout (WSSA 1983) (Table E6-16). Toxicity values for amphibians, aquatic invertebrates, and other fish were not found.

Metsulfuron Methyl

Metsulfuron methyl has a low toxicity for fish and aquatic invertebrates (Table E6-16). The 96-hour LC_{50} for trout and bluegill is greater than 150 mg/L (Du Pont 1984b). The 48-hour LC_{50} for the daphnia *Daphnia magna*, is also greater than 150 ppm (Du Pont 1984b). Toxicity values for amphibians and other aquatic species were not found in the literature.

Picloram

Tordon 101 (a mixture of picloram and 2,4-D) is slightly toxic, and picloram is generally moderately to slightly toxic to aquatic organisms. All reported $LC_{50}s$ for Tordon 101 are greater than 10 ppm (Table E6-24).

Aquatic insects and crustaceans have 24- to 96-hour $LC_{50}s$ of greater than 25 ppm for technical picloram. A 48-hour LC_{50} of 50.7 ppm has been reported for *Daphnia magna* exposed to technical picloram (Mayes and Dill 1984). *Daphnia* sp. showed no effect during a 24-hour exposure to 380 ppm of Tordon 101 (USDA 1984). For lake trout and cutthroat trout, technical grade picloram (90-percent active ingredient) is more toxic than the other formulations, with 96-hour $LC_{50}s$ in these species of 4.3 and 4.8 ppm, respectively (Johnson and Finley 1980).

Woodward (1979) reported increased fry mortality in cutthroat trout at concentrations of picloram (technical grade) greater than 1.3 ppm and reduced fry growth above 0.61 ppm (flow-through tests). No adverse effects to cutthroat fry occurred below 0.29 ppm. The reported concentrations are initial peak concentrations, which are intended to simulate concentration resulting from runoff from a rainstorm. Mean concentrations for the exposure period were not reported. Similar findings have been reported by Scott et al. (1977, as cited in Mullison 1985). Woodward (1976) has also reported chronic studies on lake trout, where 0.035 ppm of picloram adversely affected the rate of yolk sac absorption and growth of fry.

Mayes et al. (1987) conducted chronic toxicity studies with embryo-larval rainbow trout exposed to technical picloram. They reported an MATC of between 0.55 ppm and 0.88 ppm and estimated as 0.70 ppm based on the geometric mean. Larval survival was significantly reduced at 2.02 ppm, and growth was significantly reduced at 0.88 ppm.

No adverse effects on growth were reported for algae, *Daphnia* sp., goldfish, and guppies exposed to 1 ppm picloram for 10 weeks. Guppies exhibited no adverse effects at this same concentration after 6 months of exposure

Table E6-22. Toxicity of Hexazinone to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	320-420[b]	96-hr LC_{50}	EPA 1982[a]; Du Pont 1984a
	>180[c]	96-hr LC_{50}	Mayer and Ellersieck 1986
Brook trout	>100[b,c]	96-hr LC_{50}	Mayer and Ellersieck 1986
Bluegill	505 (450-538)	96-hr LC_{50} (95% C.L.)	EPA 1982[a]
	370-420	96-hr LC_{50}	EPA 1982[a]
	925 (782-1,049)[d]	96-hr LC_{50} (95% C.L.)	Schneider and Kaplan 1983[a]
Fathead minnow	274 (270-361)[b]	96-hr LC_{50} (95% C.L.)	EPA 1982[a]; Du Pont 1984a
Fiddler crab	>1,000[b]	96-hr LC_{50}	EPA 1982[a]
Grass shrimp	56-100[b]	96-hour LC_{50}	EPA 1982[a]
Water flea Daphnia magna	151.6 (125.2-172.8)[b]	48-hr LC_{50} (95% C.L.)	EPA 1982[a]
Daphnia sp.	20-50[b] 10[b]	21-day LC_{50} 21-day NOEL	Mayack et al. 1982[a] and EPA 1982[a]
Eastern oyster -larvae	320-560[b]	48-hr EC_{50}, based on reduction in number of normal embryos.	EPA 1982[a]

[a]As cited in USDA 1984.
[b]Technical.
[c]90% wettable powder.
[d]Velpar® L - 25% hexazinone liquid.

Table E6-23. Toxicity of Imazapyr to Aquatic Organisms

Species	Concentration (ppm)	Effect
Rainbow trout	110[a] >100[b]	96-hr LC_{50}
Bluegill	>180[a] >100[b] >1,000[c]	96-hr LC_{50}
Channel catfish	>100[b]	96-hr LC_{50}
Water flea _D. magna_	>350[a] 100[b] 750[c]	48-hr LC_{50}

[a]Arsenal 2.0 AS.
[b]Technical imazapyr.
[c]Isopropylamine salt of imazapyr.
Source: American Cyanamid Company 1985.

Table E6-24. Toxicity of Picloram to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Tordon 101[e] (10.2% picloram-TIPA[a], 5.7% a.e.[b], and 21.2% a.e. 2,4-D TIPA):			
Rainbow trout	8.6 a.e. Tordon 31.8 a.e. 2,4-D	96-hr LC$_{50}$	Lynn 1965 and Winston 1963[c]
Brook trout	13.7 a.e. Tordon 50.9 a.e. 2,4-D	96-hr LC$_{50}$	Lynn 1965 and Winston 1963[c]
Brown trout	13.1 a.e. Tordon 48.8 a.e. 2,4-D	96-hr LC$_{50}$	Lynn 1965 and Winston 1963[c]
Coho salmon	17.5	24-hr LC$_{50}$	Sephar et al. 1981[d]
Green sunfish	8.6 a.e. Tordon 31.8 a.e. 2,4-D	96-hr LC$_{50}$	Kenaga 1969
Fathead minnow	3.7 a.e. Tordon 13.7 a.e. 2,4-D	96-hr LC$_{50}$	Lynn 1965 and Winston 1963[c]
Pugnose minnow	7.6 a.e. Tordon 28.2 a.e. 2,4-D	96-hr LC$_{50}$	Kenaga 1969
Goldfish	4.3 a.e. Tordon 15.9 a.e 2,4-D	24-hr LC$_{50}$	Hardy 1963[c]
Water flea *Daphnia* sp.	530	95% mortality at 24 hours; no mortality at 380 ppm.	Lynn 1965
Snail	530	100% mortality at 72 hours; no mortality at 380 ppm.	Lynn 1965
Picloram:			
Rainbow trout	24-34 (a.e.)	24 to 96-hr LC$_{50}$	DOI 1965[c]
Coho salmon	21-29 (a.e.)	96-hr LC$_{50}$	Bond et al. 1967[c]
Bluegill	21-26.5 (a.e.)	96-hr LC$_{50}$	Bond et. al. 1967[c]
Largemouth bass	13.1-19.7 (a.e.)	24 to 48-hr LC$_{50}$	DOI 1964[c]
Goldfish	14-36	24 to 96-hr LC$_{50}$	DOI 1964[c]
Mosquito fish	120-133 (a.e.)	24 to 96-hr LC$_{50}$	Johnson 1978[d]
Brown shrimp	1	48-hr NOEL	DOI 1966[d]
Water flea *Daphnia* sp.	530	95% mortality at 24 hours, NOEL at 380 ppm.	Lynn 1965
	1	No observed effect on growth and reproduction after 10 weeks.	Hardy 1966[d]

Table E6-24. Toxicity of Picloram to Aquatic Organisms (continued)

Species	Concentration (ppm)	Effect	Source
Scud Gammarus lacustris	50	24-hr LC$_{50}$	Sanders 1969[d]
	48	48-hr LC$_{50}$	DOI 1968[e]
Stonefly nymphs Pteronarcys californica	120	24-hr LC$_{50}$	Sanders and Cope 1968
Eastern oyster	1	No observed effect on shell growth after 48 hours.	Butler 1965[d]
Technical Grade (90% a.i.):			
Rainbow trout	12.5	96-hr LC$_{50}$	Johnson and Finley 1980
Lake trout	4.25	96-hr LC$_{50}$	Woodward 1976 Johnson and Finley 1980
	0.035	Decreased rate of yolk sac absorption and growth in fry, chronic exposure.	
Cutthroat trout	5.0	96-hr LC$_{50}$	Woodward 1976
	>1.3	After 22 days exposure increased fry mortality;	Woodward 1979
	>0.610 0.29	reduced growth of fry; no adverse effects.	
Bluegill	23.0	96-hr LC$_{50}$	Johnson and Finley 1980
Channel catfish	6.3-15.5	96-hr LC$_{50}$	Johnson and Finley 1980
Scud Gammarus fasciatus	0.027	96-hr LC$_{50}$	Johnson and Finley 1980
Stoneflies Pteronarcella badia	>10.0	96-hr LC$_{50}$	Johnson and Finley 1980
Pteronarcys californica	0.048	96-hr LC$_{50}$	Johnson and Finley 1980

[a]TIPA - triisopropylamine salt.
[b]a.e. - acid equivalent.
[c]As cited in Kenaga 1969.
[d]As cited in USDA 1984.
[e]As cited in Pimentel 1971.

(Lynn 1965, as cited in Ghassemi et al. 1981). Chronic studies with *Daphnia magna* by Gersich et al. (1985) indicated an MATC of between 11.8 an 18.1 ppm with a geometric mean of 14.6 ppm. The MATC endpoint was based on mean total young/adult.

Studies with picloram (Tordon 50-D) have reported 96-hour $LC_{50}s$ for 1-week-old tadpoles of 95 ppm for *Adelotus brevis* and 105 ppm for *Limnodynastes peroni* (Johnson 1976).

Simazine

Simazine is moderately to slightly toxic to fish and aquatic invertebrates (Table E6-25). Studies with the fathead minnow indicated that a 4-percent granular formulation was much more toxic than the technical material or 80 percent wettable powder. Insects and some microcrustaceans (water fleas and seed shrimp) are more sensitive than the other organisms that have been tested. No toxicity studies with amphibians are available.

Sulfometuron Methyl

Acute toxicity tests using technical sulfometuron methyl were conducted with representative aquatic species, including bluegill, rainbow trout, crayfish, and *Daphnia magna* (Table E6-26). The results indicate that this herbicide is only slightly toxic to aquatic organisms.

The fathead minnow was used for early lifestage aquatic toxicity testing. No effect on embryo hatch or larval survival and growth was observed at concentrations of up to 1.2 mg/L (Du Pont 1983).

The toxicity of sulfometuron methyl to amphibians has not been reported in the literature. No long-term studies of the effects of sulfometuron methyl on aquatic organisms have been reported.

Tebuthiuron

The toxicity of tebuthiuron to aquatic organisms is summarized in Table E6-27. This herbicide is practically nontoxic to most fish and invertebrates. Acute toxicity values are greater than 100 ppm for all aquatic species tested with the exception of the pink shrimp (96-hour $LC_{50} = 48$ ppm). Based on early life-stage studies, NOELs of 26 ppm have been determined for rainbow trout, 9.3 ppm for fathead minnow, and 21.8 ppm for *Daphnia magna*. No studies are available on amphibians.

Triclopyr

The toxicity of triclopyr to aquatic species is summarized in Table E6-28. The butoxyethyl ester is highly toxic to fish, whereas the triethylamine (TEA) salt is practically nontoxic. The 96-hour LC_{50} for bluegill exposed to the butoxyethyl ester is 0.87 ppm and is 891 ppm for exposure to the triethylamine salt. Unformulated triclopyr also is practically nontoxic to aquatic organisms. An 8-day embryo-larval study with fathead minnows exposed to the TEA salt formulation determined an MATC of 91 ppm based on mortality (Mayes et al. 1984). The hatchability of the embryos, development, and growth of the fry were not significantly affected. No toxicity studies have been reported with amphibians.

Light Fuel Oil

Diesel fuel, jet fuels, and fuel oils are moderately to highly toxic to fish (Table E6-29). Jenkins et al. (1977, as cited in Burks 1982) studied the acute and chronic toxicity of jet fuels to several fish species, including the golden shiner, rainbow trout, and flagfish. The 96-hour $LC_{50}s$ (static tests) for the golden shiner were 0.68 and 0.94 ppm for the jet fuels RJ-4 (a 12-carbon molecule) and RJ-5 (a 14-carbon molecule), respectively. The 97-day nonlethal concentration for rainbow trout was less than 0.03 ppm for RJ-4 and 0.04 ppm for RJ-5. The NOEL for eggs of the flagfish exposed by continuous flow to RJ-4 was 0.2 ppm. Reduced hatchability of flagfish was observed from exposure to RJ-5 at concentrations above 0.05 ppm.

EPA has reported (1976, as cited in DOE 1983) acute toxicity values (96-hour $LC_{50}s$) for freshwater fish of greater than 0.19 ppm for diesel fuel and greater than 1.2 ppm for No. 2 fuel oil. Tagatz (1961, as cited in Burks 1982) reported a 48-hour LC_{50} for No. 2 fuel oil of 125 to 251 ppm with juvenile American shad. This reported concentration is based on the amount of oil applied to the water's surface (nominal concentration) and not the water-soluble fraction. This may account for the apparent lower sensitivity of the shad to No. 2 fuel oil.

The toxicity of No. 2 fuel oil has been studied for many different marine fish and invertebrate species (Table E6-29). The $LC_{50}s$ range from 0.81 to greater than 6.9 ppm for marine fish and 0.21 to 14.1 ppm for invertebrates (Connell and Miller 1984). The range of toxicity values determined for No. 2 fuel oil with marine species is useful in estimating the range of sensitivities for freshwater species because marine and freshwater species generally have a similar range of tolerance to toxicants (Sprague 1985).

Irwin (1964, as cited in Burks 1982) calculated a "ratio of resistance" to rank the sensitivities of 57 fish species to oil refinery wastewater. The guppy was the least sensitive and was assigned a ratio

Table E6-25. Toxicity of Simazine to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	>100 (technical)	96-hr LC$_{50}$	Mayer and Ellersieck 1986
	2.5 (dissolved in 0.1% acetone)	No effects after 28 days exposure and 56 days of observation.	Bathe et al. 1975[a]
	2.8	96-hr LC$_{50}$	Mauck 1974[a]
Bluegill	100 (90.9 - 110) (80% wettable powder)	96-hr LC$_{50}$ (95% C.L.)	Mayer and Ellersieck 1986
	11.6	96-hr LC$_{50}$	Mauck 1974[a]
	36 (simazine 80W	96-hr LC$_{50}$	Mauck 1974[a]
Fathead minnow	>10 (technical)	96-hr LC$_{50}$	Mayer and Ellersieck 1986
	510 (373 - 698) (80% wettable powder)	96-hr LC$_{50}$	
	5 (3.5 - 7.15) (4% granular)	96-hr LC$_{50}$	
Channel catfish	85 (simazine 80W)	96-hr LC$_{50}$	Mauck 1974[a]
Pumpkinseed	28 (simazine 80W)	96-hr LC$_{50}$	Mauck 1974[a]
Redear sunfish	55 (simazine 80W)	96-hr LC$_{50}$	Mauck 1974[a]
Largemouth bass	25-45 (simazine 80W)	96-hr LC$_{50}$	Mauck 1974[a]
Yellow perch	100 (simazine 80W)	96-hr LC$_{50}$	Mauck 1974[a]
Bluntnose minnow	63 (simazine 80W)	96-hr LC$_{50}$	Mauck 1974[a]
Scud *Gammarus fasciatus*	130 (technical)	96-hr LC$_{50}$	Mayer and Ellersieck 1986
G. lacustris	13	96-hr LC$_{50}$	USDA 1984

Table E6-25. Toxicity of Simazine to Aquatic Organisms (continued)

Species	Concentration (ppm)	Effect	Source
Seed shrimp *Cypridopsis vidua*	3.7 (2.6 - 5.3)	48-hr LC_{50}	Mayer and Ellersieck 1986
Grass shrimp *Palaemonetes*	>5.6	24-hr LC_{50}	Mayer and Ellersieck 1986
Stonefly *Pteronarcys californica*	1.9 (0.9 - 4.0) (technical)	96-hr LC_{50}	Mayer and Ellersieck 1986
Water flea *Daphnia magna*	1.1 (0.56 - 2.2) (technical)	48-hr EC_{50}	Johnson and Finley 1980
Sowbug *Asellus brevicaudus*	>100 (technical)	48-hr LC_{50}	Sanders 1970[a]
Crayfish *Orconectes nais*	>100	48-hr LC_{50}	Sanders 1970[a]

[a]As cited in USDA 1984.

Table E6-26. Toxicity of Sulfometuron Methyl to Aquatic Organisms

Species	Concentration (ppm)	Effect
Rainbow trout	>12.5[a]	96-hr LC_{50}
Bluegill	>12.5[a]	96-hr LC_{50}
Fathead minnow	1.2 No effect on eggs or larvae.	
Crayfish	>5,000[b]	96-hr LC_{50}
Water flea *D. magna*	>12.5[a] 8,500[c]	48-hr LC_{50} 48-hr EC_{50}

[a]This represents the limits of solubility for the technical product under the reported test conditions.
[b]Technical product; experimental conditions (pH) were adjusted to increase solubility.
[c]75% dry flowable formulation.

Source: Du Pont 1983; Fred O'Neal, Du Pont, Agricultural Products Department, Wilmington, Delaware, personal communication, 1987.

Table E6-27. Toxicity of Tebuthiuron to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	144	96-hr LC_{50}	USDA 1986
- eggs & larvae	26	NOEL, no effects on hatchability, growth, behavior development, or survival; reduced growth and survival at 52 ppm.	USDA 1986
Bluegill	112	96-hr LC_{50}	USDA 1986
Fathead minnow	>160 (technical)	96-hr LC_{50}	Todd et al. 1974[a]
- eggs & larvae	9.3	NOEL, no effects on hatching, growth, development, behavior, or survival; reduced growth at 18 ppm.	USDA 1986
Goldfish	>160	96-hr LC_{50}	Todd et al. 1974[a]
Fiddler crab	>320	96-hr LC_{50}, 320 ppm was highest concentration tested.	USDA 1986
Pink shrimp	48	96-hr LC_{50}	USDA 1986
Water flea Daphnia magna	297	48-hr EC_{50}	USDA 1986
	21.8	No effects on reproduction, growth, or survival with lifetime exposure.	USDA 1986
Oyster			
- embryos	180-320	48-hr EC_{50}, abnormal development.	USDA 1986

[a]As cited in USDA 1986.

Table E6-28.   Toxicity of Triclopyr to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	0.74[a]	96-hr LC_{50}	Dow Chemical Company 1983[b]
	552[c]	96-hr LC_{50}	
Bluegill	117	96-hr LC_{50}	Dow Chemical Company 1983[b]
	891[c]	96-hr LC_{50}	
	148 (triclopyr)	96-hr LC_{50}	
Fathead minnow	120 to 245[c,d]	96-hr LC_{50} (Toxicity increased with temperature between 17 and 26 °C)	Mayes et al. (in press)[b]
	101 (88.5 - 116)[c,d]	8-day LC_{50} (24.8 - 25.8 °C)	
- embryo-larval stages	114[c,d]	98% mortality at 28 days; 21% mortality in controls.	
Crab	>1,000[e]	96-hr LC_{50}	Dow Chemical Company 1983[b]
Shrimp	895[e]	96-hr LC_{50}	Dow Chemical Company 1983[b]
Water flea *Daphnia magna*	1,170 (1,030 - 1,340)[c]	48-hr LC_{50} (95% C.L.)	Gersich et al. 1984
	1,140 (950 - 1,590)[c]	21-day LC_{50}	
	110	MATC based on total young and brood size.	
Oyster	56 - 87[e]	48-hr LC_{50}	Dow Chemical Company 1983[b]

[a] Garlon® 3A butoxyethyl ester.
[b] As cited in USDA 1984.
[c] Garlon® 3A-triethylamine salt (TEA) or other TEA formulation.
[d] Flow-through tests.
[e] Garlon® 3A-type not specified.

Table E6-29. Toxicity of Light Fuel Oil to Aquatic Organisms

Species	Concentration (ppm)	Effect	Source
Freshwater fish	>0.19[a]	96-hr LC_{50}	EPA 1976, as cited in DOE 1983
	>1.2[d]	96-hr LC_{50}	
Rainbow trout	0.03[b]	97-day nonlethal level	Jenkins et al. 1977, as cited in Burks 1982
	0.04[c]	97-day nonlethal level	
Dolly Varden trout smolts	2.29[d]	96-hr LC_{50}	Connell and Miller 1984
Pink salmon	0.81[d]	96-hr LC_{50}	Connell and Miller 1984
Golden shiner	0.68[b]	96-hr LC_{50}	Jenkins et al. 1977, as cited in Burks 1982
	0.94[c]	96-hr LC_{50}	
Sheepshead minnow	>6.9[d]	96-hr LC_{50}	Connell and Miller 1984
Saffron cod	2.93[d]	96-hr LC_{50}	Connell and Miller 1984
Flagfish (eggs)	0.2[b]	No effect level.	Jenkins et al. 1977, as cited in Burks 1982
	>0.05[c]	Reduced hatchability	
Blue crab	14.1[d]	96-hr LC_{50}	Melzian 1983
Grass shrimp		96-hr LC_{50}	Connell and Miller 1984
larvae	1.2[d]		
post larvae	2.4[d]		
adult	3.5[d]		
Brown shrimp		96-hr LC_{50}	Connell and Miller 1984
late juvenile	2.9[d]		
adult	4.9[d]		
Dark shrimp	1.11[d]	96-hr LC_{50}	Connell and Miller 1984
Humpback shrimp	1.69[d]	96-hr LC_{50}	Connell and Miller 1984
Scooter shrimp	0.53[d]	96-hr LC_{50}	Connell and Miller 1984
Pink shrimp	0.21[d]	96-hr LC_{50}	Connell and Miller 1984
Polychaete (segmented aquatic worm)	2 - 4.2[d]	96-hr LC_{50}	Connell and Miller 1984

[a] Diesel fuel.
[b] Jet fuel RJ-4.
[c] Jet fuel RJ-5.
[d] No. 2 fuel oil.

of resistance of 100. The ratios of resistance for some common freshwater fish were as follows: rainbow trout (34.68), smallmouth bass (35.60), northern pike (37.31), fathead minnow (49.19), largemouth bass (53.27), bluegill (54.10), and channel catfish (60.15). This study may be useful in predicting the relative order of sensitivities of these species to diesel fuels and other petroleum products.

The 96-hour LC_{50} for adult blue crabs exposed to No. 2 fuel oil was 14.1 ppm (Melzian 1983). This species appears to be much more tolerant than other crustaceans or fish tested. No histopathological changes were observed in the gills, hepatopancreas, or muscles of the blue crab after 2 weeks of exposure to No. 2 fuel oil at 0 to 1.0 ppm (Melzian 1983).

A spill of No. 2 fuel oil into a small stream in Virginia was acutely toxic to some fish, crayfish, and caddisflies (order Trichoptera) (Hoehn et al. 1974, as cited in Burks 1982). Two weeks after the spill, the density of benthic macroinvertebrates downstream was 25 percent less than the density upstream from the spill, but species diversity was not affected. The density of the macroinvertebrates returned to normal levels by 18 weeks after the spill.

The toxicity of diesel fuel or other related petroleum compounds to amphibians has not been reported in the literature. No chronic toxicity studies have been reported for any aquatic organisms.

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Section E7
Nontarget Species
Exposure Analysis

This section describes the estimated wildlife and aquatic species exposures to the 19 herbicides and 2 carriers used in vegetation treatment in the 13 Western States. It discusses the representative species selected for exposure estimation and presents details about how exposures for each species were determined, based on the species biology and chemical application rates.

Wildlife Exposures

Representative Wildlife Species

Wildlife exposures were calculated for a group of wildlife species representative of those typically found in areas supporting rangeland vegetation in the West. These species represent a range of phylogenetic classes and body sizes, with many different diets. The methodology used to determine the exposures is the same as that used in the environmental impact statements prepared by the U.S. Department of Justice, Drug Enforcement Administration, on the eradication of cannabis with herbicides (DEA 1985, 1986), and the environmental impact statement prepared by the U.S. Department of the Interior, Bureau of Land Management, on the control of noxious weeds with herbicides (USDI 1987). Table E7-1 lists the representative wildlife species and gives the various biological parameters used for each species in the exposure analysis.

Wildlife Data Sources

The following references were used in the species selection and in deriving the biological parameters of each species:

(1) Distribution, Life History, and Diet
  • Birds: Robbins et al. (1966), Scott et al. (1977), Brown (1985), MacMahon (1986).
  • Mammals: Schmidt and Gilbert (1978), Burt and Grossenheider (1966), Whitaker (1962), Young and Jackson (1951), USDA (1935), Brown (1985), MacMahon (1986), McCracken (1981).

  • Reptiles and Amphibians: Conant (1958), Dickerson (1969), Brown (1985), MacMahon (1986).

(2) Physiology, Metabolism, Food Intake, and Weight
  • Gordon et al. (1968), Hutchinson et al. (1968), Lasiewski and Dawson (1967), Kendeigh (1970), Lasiewski and Calder (1971), Schmidt-Nielsen (1975), Schmidt-Nielsen (1972), Sturkie (1965), Slobodkin and Richman (1961), Welty (1962), Zar (1968), Drozdz (1968), Odum (1971), Moore (1964), Altman and Dittmer (1962), Seibert (1949), Banse and Mosher (1980), Odum et al. (1962), Damuth (1981), Kendeigh (1969), Weins and Innis (1974), Rosenberry and Klimstra (1971).

Wildlife Exposure Estimates

Realistic and extreme acute exposure estimates were made for each representative species for each of the three major exposure routes: inhalation, dermal, and ingestion. Because the herbicides degrade relatively rapidly and sites are normally treated only once per year, no analysis of chronic wildlife dosing was done. Because the herbicides show little tendency to bioaccumulate, as discussed in Chapter 3 of the EIS, long-term persistence in food chains and subsequent toxic effects were not considered a problem and were not examined in the risk analysis.

Herbicide doses for the representative species were calculated using conservative, simplified assumptions concerning routine application operations that give realistic dose estimates and highly unlikely (extreme) dose estimates in which animals are directly sprayed with herbicide. Exposures for rangeland typical applications were based on the typical application rates for aerial rangeland. Typical and extreme exposures were estimated for right-of-way applications.

For the rangeland typical doses, dermal exposures were based on the levels likely to be found on vegetation leaf surfaces, because the animals are assumed to seek cover during a spraying operation. The right-of-way typical dose levels were estimated the same way but

Table E7-1. Biological Parameters of Representative Rangeland Wildlife Species

Representative Species	Body Weight (grams)	Body Surface Area (cm²)	Vegetation Contact (percent)	Percent Body Groomed	Breathing Rate (L/min)
Birds					
Lark Bunting	33	103	77	62	2.05×10^{-2}
Sage Grouse	1,500	1,314	18	21	0.39
Bobwhite Quail	170	307	42	39	7.26×10^{-2}
American Kestrel	125	250	47	42	5.73×10^{-2}
Mammals					
Grasshopper Mouse	32	101	78	63	2.41×10^{-2}
Black-Tailed Jackrabbit	2,722	1,955	15	17	0.84
Pronghorn	46,100	12,903	5	8	8.12
Coyote	15,500	6,237	8	10	3.40
Reptiles					
Horned Lizard	25	86	86	0	0.00
Yellow-Bellied Racer	395	539	30	0	0.00
Amphibians					
Rocky Mountain Toad	22	79	90	0	0.01
Domestic Animals					
Beef Cow	453,590	59,292	2	4	50.58
Chicken	2,000	—	—	—	—
Domestic Dog	13,000	—	—	—	—

at the higher rates used in those applications. Extreme doses were estimated by assuming that animals are directly sprayed at the right-of-way application rate.

The dermal penetration rates used in the human exposure analysis were used to determine mammalian wildlife dermal penetration (that is, the amount of chemical that penetrates an animal's skin). This is a conservative assumption because the animal's fur is likely to decrease the amount of herbicide that actually reaches the skin. A dermal penetration rate of 10 percent was assumed for the herbicides for which no dermal penetration data were available. In both typical and extreme exposures, mammals and birds are assumed to receive an oral dose from grooming their fur or preening their feathers. This amount is subtracted from the amount they would receive from their dermal exposure.

Because larger animals have larger home ranges, they are not as likely to feed on contaminated items at a particular site as are smaller animals. Therefore, ingestion doses

were assumed to come from animals eating a specified percentage of their daily food intake in contaminated items, based on their body size. That is, the percentage of contaminated food intake decreases as body size increases.

Inhalation exposures are assumed to come from a hypothetical amount of herbicide droplets forming a "cloud" that moves slowly offsite.

The total systemic dose to each animal was calculated as the sum of the estimated doses received by way of dermal, ingestion, and inhalation routes. Tables E8-1 to E8-22 in the Wildlife Risk Analysis Section (Section E8) give the total typical and extreme dose estimates for the representative species.

Exposure Calculations

Inhalation Exposures. Wildlife inhalation exposures were assumed to come from animals breathing in herbicide spray droplets of respirable size (30 microns in diameter or less) as a hypothetical "cloud" of those droplets moves slowly offsite. The cloud is

assumed to be dispersed within the first 5 meters above ground level and to consist of respirable droplets that constitute 1 percent of the total applied herbicide by volume. Based on these assumptions, the airborne concentration is 0.0002242 milligram per liter (mg/L) for each 1.12 kilogram per hectare (kg/ha) (1 lb/acre) applied. The cloud moves offsite and dissipates, exposing animals on the downwind edge for 10 minutes. The nominal exposure was multiplied by the herbicide application rate and then by each animal's breathing rate. Their breathing rate in liters per minute is based on the following equations:

$$\text{Birds: LPM} = \frac{284 \times (BWT/1,000)^{.77}}{1,000}$$

$$\text{Mammals: LPM} = \frac{379 \times (BWT/1,000)^{.80}}{1,000}$$

Reptiles: LPM = 0.00334

Amphibians: LPM = 0.007

where:

LPM is the animal's breathing rate in liters per minute

BWT is the animal's body weight in grams

The equations for birds and mammals were taken from Lasiewski and Calder (1971). The reptile value is from Gordon et al. (1968), who report a study on the collared lizard. The breathing rate for amphibians was from Hutchinson et al. (1968). As anticipated, the animal modeling results showed inhalation exposures to be only a small fraction of each species' total dose.

Dermal Exposures. Dermal exposures are assumed to come from two sources: (1) directly from herbicide spray at the deposition rate that should occur on vegetation leaf surfaces in the typical case and at the herbicide application rate in the extreme case, and (2) indirectly by contact with contaminated vegetation.

Fur, feathers, and scales afford varying degrees of protection against dermal exposure; by preventing a chemical from reaching an animal's skin, they may allow the chemical to dry or to be rubbed off during movement. For this reason, the dermal penetration rate for

each herbicide for mammals was adjusted for three other animal classes—birds, reptiles, and amphibians. Dermal penetration factors were multiplied by the mammalian penetration rate as follows: (1) birds, 0.75; (2) reptiles, 0.15; and (3) amphibians, 5.0. The amphibian factor is high because the moist, glandular skin of the amphibian serves to a large extent as a respiratory organ and is much more permeable than the skin of the other animal classes (30 percent (range of 5 to 93 percent) of body weight in water moves through skin in 24 hours according to Moore (1964)).

Wildlife may receive indirect dermal exposure by brushing against contaminated vegetation. The amount transferred would depend on (1) the density of the vegetation, (2) the animal's body size in relation to the height of the vegetation, and (3) the amount of the animal's movement.

To simplify the analysis, it was assumed that a certain percentage of the animal's total body surface received herbicide at the same level as the direct dermal exposure (either the level on leaf surfaces in the realistic case or at the application rate in the extreme case). That percentage was based on the animal's body size and a movement factor (MVF) to adjust for the taxonomic class (mammals, for example, are expected to move more than amphibians). The animal's total body surface area was assumed to be a function of its weight according to the following formula (Kendeigh 1970, Schmidt-Nielsen 1972):

$$BSA = 10 \times (BWT)^{.667}$$

where:

BSA is the animal's body surface area in square centimeters (cm^2)

BWT is the animal's body weight in grams

The animal's vegetation contact percent (VCP) is based on its body weight in grams (BWT) according to the following formula:

$$VCP = 2.89(BWT)^{-0.10526}$$

The class adjustment factors (MVFs) for differing movement are as follows: (1) birds, 0.8; (2) mammals, 1; (3) reptiles, 0.3; and (4) amphibians, 0.4. The indirect dermal dose (IND) is then calculated using the direct dermal dose (DDD):

$$IND = DDD \times VCP \times MVF$$

Mammals and birds groom themselves regularly and may receive an ingestion dose if their fur or feathers are contaminated. The percentage of their body surface groomed (PBG) was assumed to be a decreasing function of their body size according to the following formula:

$$PBG = 1.72 \ (BWT)^{-0.29}$$

No grooming was assumed for reptiles and amphibians. The oral dose for mammals and birds from grooming was subtracted from the amount of herbicide that would contribute to the animal's dermal dose.

Therefore, the typical dermal dose was calculated as follows:

Typical dermal dose (mg/kg) = {[DDD(mg) + IND(mg)] x, (1.00−PBG) x dermal penetration rate}/BWT(kg)

These equations are mathematical expressions of the relationship assumed by the preparers of this risk assessment between body weight and body size, and body weight and metabolic parameters.

Ingestion Doses. Each representative species was assumed to feed on contaminated food items according to a specified diet and to drink a specified amount of water. These dietary amounts are listed in Table E7-2. Diets may vary from season to season and across the species range; the diet items and amounts were chosen to be a reasonable representation of what an individual animal might consume on a given day. The diet items—grass, forage vegetation, seeds, and insects—are assumed to have the following contamination levels in ppm from ground application based on field studies by Hoerger and Kenaga (1972) for a 1-lb/acre application rate:

Diet Item	Residue (ppm)
Grass	92
Forage	33
Seeds	3.2
Insects	4.8

In the typical case, animals are assumed to drink water from a stream offsite that reaches a concentration of 0.001267 ppm per pound of herbicide applied per acre for aerially applied

herbicides and 0.0003 ppm for ground-applied herbicides. In the extreme case, water reaches a concentration of 0.0068 ppm for aerially applied herbicides and 0.00063 ppm for ground-applied herbicides. Predators that feed on mice, toads, or quail are assumed to receive the total body burden that each of these prey species has received through the three exposure routes described above as a result of the herbicide spraying operation. Each species is assumed to consume a percentage of its daily intake in contaminated food items, depending on its body size. The percentages of food contaminated (PFC) (listed in Table E7-1) are based on the following formula:

$$PFC = 100 \ x \ (1/BWT)^{0.2}$$

The total ingestion dose is the sum of the doses received from contaminated food, contaminated water, and from grooming.

Aquatic Species Exposures

Representative Aquatic Species

Representative species typical of aquatic habitats in the West are given in Table E8-23 in the next section of this Appendix. These species were assumed to be exposed by immersion to estimated concentrations of the 19 herbicides and 2 carriers in bodies of water with specified characteristics.

Aquatic Exposure Estimates

Exposure was assumed to occur for herbicides that drift offsite from aerial rangeland and right-of-way applications. Typical estimated environmental concentrations (EECs) of each herbicide were computed for a body of water one-fourth acre in area and 4 feet deep. Typical EECs were based on typical application rates and a distance of 200 feet from the application site to the body of water. EECs for kerosene and diesel oil were based on the amount of kerosene and diesel oil used in application of herbicide ester formulations.

To assess the effects of accidents, aquatic EECs were calculated for a direct spray at the highest application rate of herbicide into a pond and a spill of an 80-gallon helicopter load of herbicide mixture into a pond. In all cases, the spill into the pond results in higher EECs than a direct spray. The exposure levels from the typical and accident EECs are described in Section E8 in the aquatic species risk analysis.

Table E7-2. Representative Wildlife Species Daily Diet Items

Representative Species	Water	Grass	Seeds	Insects	Grass-hoppers	Mouse	Toad	Quail
Birds								
Lark Bunting	0.02	—	1	2	6	—	—	—
Sage Grouse	0.10	—	—	30	40	—	—	—
Bobwhite Quail	0.05	—	4	10	20	—	—	—
American Kestrel	0.05	—	—	—	20	32	—	—
Mammals								
Grasshopper Mouse	0.01	1	1	1	6	—	—	—
Black-Tailed Jackrabbit	0.05	300	—	—	—	—	—	—
Pronghorn	1	2,763	—	—	—	—	—	—
Beef-Cow	58	11,250	—	—	—	—	—	—
Coyote	0.80	—	—	—	40	320	—	340
Reptiles								
Horned Lizard	0.01	—	—	3	5	—	—	—
Yellow-Bellied Racer	0.01	—	—	3	6	—	22	—
Amphibians								
Rocky Mountain Toad	0.05	—	—	2	2	—	—	—

Note: Water amount in liters; food items in grams.

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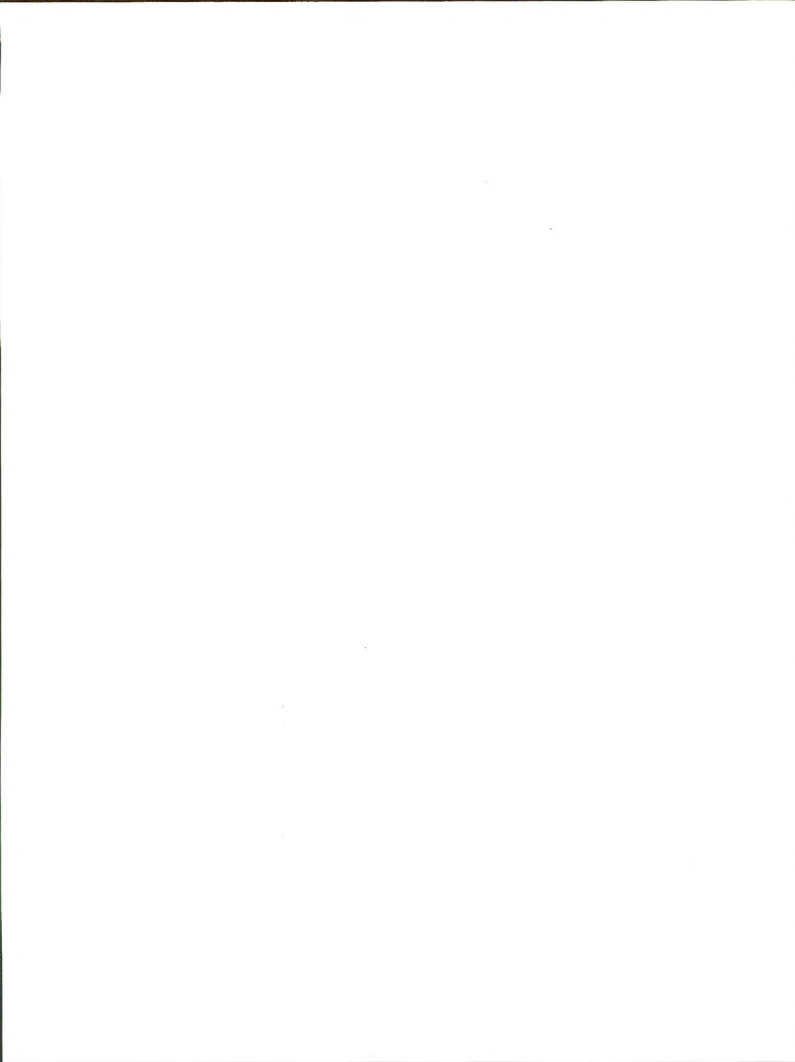
Section E8
Nontarget Species Risk Analysis

The risk analysis considers potential wildlife and aquatic species impacts of using 19 herbicides in the BLM vegetation treatment program. Wildlife and aquatic species risk from vegetation management with herbicides is a function of the inherent toxicity (hazard) of each herbicide to different organisms and of the amount of each chemical (exposure) those organisms may take in as a result of a vegetation management operation. The wildlife and aquatic species risk analysis compares estimated acute exposures of representative species determined in the previous section with acute toxicity levels found in laboratory studies. Common and scientific names for the representative species are listed in Appendix F.

Wildlife Risk Analysis

Wildlife Risk Analysis Criteria

For wildlife risks, the criteria used by the Environmental Protection Agency (EPA) in ecological risk assessment (EPA 1986) were used to judge the absolute risks to the different representative species and the relative risks among the 19 herbicides and the carriers diesel oil and kerosene. EPA criteria call for comparison of an estimated environmental concentration (EEC) with a laboratory-determined LD_{50} for the most closely related laboratory test species.

Where the EEC exceeds 1/5 LD_{50}, EPA deems it a risk that may be mitigated by restricting use of the pesticide. EPA judges EECs that exceed the LD_{50} as unacceptable risks. Doses below the 1/5 LD_{50} level are assumed to present a low or negligible risk. In this risk assessment, an organism's total estimated dose (rather than an EEC) is compared with the laboratory toxicity level because the dose comes from all exposure routes, not just feeding. Risks resulting from exposures that exceed 1/5 LD_{50} are termed "moderate," while those that exceed the LD_{50} are classified as "significant."

An analysis of the herbicide risk to wildlife compared estimated acute doses for the representative wildlife species described in Section E7 with available hazard information on the most closely related species as described in Section E6. Because the herbicides examined in this appendix show little tendency to bioaccumulate, long-term persistence in food chains and subsequent toxic effects, such as those that have resulted from the use of the persistent organochlorides, are not considered a problem and are not examined in the risk analysis. No analysis of chronic wildlife dosing was done because the herbicides degrade relatively rapidly and sites are normally treated only once in a given year.

Wildlife Toxicity Surrogates

Species that represent the wildlife commonly found in the vegetation treatment program area were identified, and the doses they would receive from exposure to the 19 herbicides and diesel oil and kerosene were calculated in Section E7. However, in some cases, information was not available on doses that would cause toxicity to these representative species. Therefore, it was necessary to select a closely related species for which toxicity data were available. For example, the coyote was selected as a common carnivorous mammal in the Western United States but, because toxicity data were not available for 2,4-D on the coyote, the dog was identified as a closely related species and data for it were used in calculations.

Surrogates for Avian and Mammalian Toxicity

Toxicity data on the most closely related avian or mammalian species are used for the wildlife risk comparisons. For avian species, mallard ducks are used only when no data on an upland species, such as the Japanese quail or pheasant, are available. Where no data on a mammalian wildlife species (for example, pronghorn) are available, data on laboratory mammals (such as rats, mice, or dogs) are used.

Surrogates for Amphibian and Reptile Toxicity

In testing nearly 200 chemicals on terrestrial vertebrate wildlife species, the U.S. Fish and Wildlife Service (Hudson et al. 1984) studied the effects of 17 pesticides on the adult bullfrog and the mallard duck. There was a good correlation ($r = 0.67$) between the LD_{50}s for the bullfrog and mallard for the tested

chemicals. The bullfrog LD_{50}s were higher in 12 of the 17 cases, often by more than an order of magnitude, allowing for reasonable and conservative use of the mallard data to estimate risk to amphibians.

The U.S. Fish and Wildlife Service also reviewed the available data on the toxicity of environmental contaminants to reptiles (Hall 1980). Most of the data consisted of residue levels of organochlorides in reptiles after field applications. There were no data of the type reported in the above amphibian studies relating dose levels to lethality; however, the author noted that avian data could serve as a guide for reptile toxicity because birds were closely related to reptiles even though, in general, reptiles appeared to be more susceptible to pesticides than birds or mammals.

Thus, for the herbicides and carriers considered for vegetation treatment in this environmental impact statement, suitable data are lacking for terrestrial stages of amphibians and for reptiles. Because there is a reasonable correlation between avian and amphibian toxicity as indicated in the mallard vs. bullfrog LD_{50} analysis and reason to suspect a similar relationship between avian and reptilian toxicity as noted by Hall (1980), available avian toxicity data were used as surrogates for both amphibians and reptiles.

Wildlife toxicity reference levels used to assess the risks of the 19 herbicides and 2 related additives are given at the end of this section in Tables E8-1 through E8-21.

Wildlife Exposure Analysis

Tables E8-1 through E8-21 give the total typical and extreme dose estimates for the 14 representative wildlife species for each of the herbicides and carriers being evaluated in this EIS. Details on the calculation of these exposures are presented in Section E7 of this appendix.

Wildlife Risk Overview

The wildlife risk assessment tends to overstate the risks because many of the assumptions are quite conservative. For example, no degradation of the herbicides is assumed to occur and all herbicide sprayed is assumed to be biologically available. Dermal exposures are assumed to come both directly from herbicide spray and indirectly from brushing up against treated vegetation. This accumulation of doses from almost every conceivable route undoubtedly overestimates doses, even in the

typical case. Nevertheless, when these dose estimates do exceed the EPA risk criterion, and more so when they exceed the LD_{50} for the most closely related laboratory species, there is a clear risk of adverse effects to individual animals.

In general, based on the available toxicity data and on the proposed application rates, risks to wildlife are low for most of the herbicides. Estimated doses for typical rangeland exposures and typical rights-of-way exposures result in a negligible risk from all herbicides considered, as well as diesel oil and kerosene. The application rates for several of the herbicides used on rights-of-way, coupled with extreme exposure estimates, present moderate risks to some species. However, the estimated exposures exceed the LD_{50} only under extreme assumptions for lark buntings during the use of atrazine. The typical dose estimates are below the EPA risk criterion of one-fifth LD_{50} and are far below the laboratory species LD_{50} in most cases.

The risks of the individual herbicides are discussed below. Literature references for the toxicity levels in laboratory species are given in the wildlife hazard analysis. Again, it must be noted that there are very few toxicity studies on which to base these conclusions. Avian toxicity data are particularly rare for most of the herbicides. Several herbicides had only two or three laboratory animal LD_{50} tests to use in the analysis. However, the conservatism used in estimating the wildlife doses should compensate for much of the uncertainty in the toxicity data base.

Wildlife Risk From the Individual Herbicides

Even using worst case assumptions, the use of amitrole, chlorsulfuron, dalapon, glyphosate, hexazinone, imazapyr, metsulfuron methyl, picloram, sulfometuron methyl, diesel oil, or kerosene is not expected to pose unacceptable risks to terrestrial wildlife.

The use of atrazine on rights-of-way presents a moderate risk of adverse effects to bobwhite quail, black-tailed jackrabbit, American kestrels, grasshopper mice, and Rocky Mountain toads for extreme exposures. Extreme exposures on rights-of-way to lark buntings result in a significant risk.

Bromacil, clopyralid, and dicamba result in moderate risks to lark buntings under extreme rights-of-way assumptions. 2,4-D presents moderate risks for the extreme right-of-way scenario to lark buntings, American kestrels,

grasshopper mice, and Rocky Mountain toads. Extreme rights-of-way exposures to diuron present moderate risks for lark buntings, grasshopper mice, and Rocky Mountain toads. Extreme rights-of-way exposures to simazine present moderate risks for lark buntings and grasshopper mice. Extreme rights-of-way exposures to tebuthiuron result in moderate risks to lark buntings, grasshopper mice, and black-tailed jackrabbits. Extreme rights-of-way exposures to triclopyr cause a moderate risk to grasshopper mice.

Local populations of small mammals, small birds, terrestrial amphibians, and reptiles may be adversely affected if large areas are treated; however, the reproductive capacity of these species is generally high enough to replace the few lost individuals within the next breeding cycle. Populations of larger mammals and birds and any domestic animals present are not likely to be affected at all.

Aquatic Risk Analysis

The risks of adverse effects from exposure to herbicides that drift offsite from aerial rangeland and rights-of-way applications were estimated for the representative aquatic species described in the previous section (see Table E7-5 in section E7 of this appendix). Risks were also estimated for an accidental direct spray of a pond and an accidental helicopter jettison of its entire load into a pond. Acute toxicity reference values (LC_{50}s or EC_{50}s) used in the analysis were selected for the representative species from the summary tables presented in the aquatic hazard analysis (Section E6).

Risks were calculated for four aquatic species on which toxicity data were generally available for the herbicides. Trout were chosen to represent cold-water fish, bluegills to represent warm-water fish, and *Daphnia* to represent aquatic invertebrate species. Risks to fathead minnows were also evaluated because toxicity information was generally available on this species.

To estimate the risk of adverse effects occurring, the selected toxicity reference values were compared to the estimated environmental concentrations of each herbicide for a body of water one-fourth acre in area and 4 feet deep. The toxicity reference levels and the EECs for each of the 19 herbicides and the 2 carriers are given in Table E8-22 at the end of this section. The ratio of the EEC to the LC_{50} (or EC_{50}) is named the quotient value (Q-value). Typical EECs were based on

typical application rates and a distance of 200 feet from the application site to the body of water. EECs for kerosene and diesel oil were based on the amount used in application of herbicide esters, such as 2,4-D and triclopyr. The Q-values were compared to the risk criteria proposed by EPA (1986), where the risks of adverse effects to fish or invertebrates are estimated as follows:

Q-value	Risk
EEC/LC_{50} <0.1	No acute risk
EEC/LC_{50} ≥0.1 and <0.5	Moderate risk; presumption of risk that may be mitigated
EEC/LC_{50} ≥0.5	Significant risk of acute effects

Aquatic Risk Overview

According to risk calculations for realistic (typical) exposures, risks to aquatic species are low for all herbicides proposed for use. The only risk identified in typical cases is a moderate risk posed by the use of kerosene as an herbicide carrier. Use of appropriate buffer strips along bodies of water and avoidance of spraying on windy days would reduce this risk. No adverse effects are expected on the aquatic ecosystem as a whole. Risks from accidental direct spray of a water body or an accidental jettison of herbicide mixture into a water body are significant, but the probability of either event occurring is low.

Drift Onto a Pond at Typical Rangeland Application Rates

In this scenario, the only risk identified is a moderate risk to trout from the use of kerosene as a carrier for herbicide esters, such as 2,4-D and triclopyr.

Drift Onto a Pond at Typical Rights-of-Way Application Rates

In this scenario, kerosene presents a moderate risk to trout.

Accidental Direct Spray of Pond at Highest Application Rate

This accident scenario presents risks to aquatic species from several herbicides. There would be moderate risks to bluegills from diuron or simazine, to *Daphnia* from dalapon, to trout and fathead minnows from atrazine, and to fathead minnows and *Daphnia*

from 2,4-D. Significant risks were identified for *Daphnia* from amitrole, atrazine, and clopyralid; for trout, fathead minnows, and *Daphnia* from simazine; and for trout, bluegills, and pink shrimp from diesel oil.

Helicopter Jettison of 80 Gallons of Mix Into a Pond

There are either moderate or significant risks to all species from most of the herbicides for a helicopter jettison into a pond. However, the probability of this type of accident is extremely low.

Potential Effects on Threatened or Endangered Species

Federal policies and procedures for protecting threatened and endangered species of fish, wildlife, and plants were established by the Endangered Species Act of 1973 (16 U.S.C. 1531 et seq.) and regulations issued pursuant to the act. The purposes of the act are to provide mechanisms for conservation of threatened and endangered species and the habitats upon which they depend, and to achieve the goals of international treaties and conventions related to endangered species. Under the act, the Secretary of the Interior is

required to determine which species are threatened or endangered and to issue regulations for the protection of those species.

Risks to endangered species will be analyzed at a site-specific level. These analyses will be based, in part, on the results of the risk estimates for the representative species examined in this risk assessment.

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EPA. U.S. Environmental Protection Agency.

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Table E8-1. Risk Comparison of Estimated Wildlife Doses From Amitrole to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	14.9	29.9	248	400	2,000	Mallard
Sage Grouse	1.02	2.04	21.5	400	2,000	Mallard
Bobwhite Quail	4.70	9.41	86.3	400	2,000	Mallard
American Kestrel	5.60	11.2	104	400	2,000	Mallard
Mammals						
Grasshopper Mouse	20.5	41.0	279	3,000	15,000	Mouse
Black-tailed Jackrabbit	6.34	12.7	43.0	2,000	10,000	Rabbit
Pronghorn	1.87	3.73	11.4	2,000	10,000	Rabbit
Coyote	0.462	0.923	7.97	350	1,750	Cat
Reptiles						
Horned Lizard	1.10	2.19	8.46	400	2,000	Mallard
Yellow-Bellied Racer	0.109	0.218	1.82	400	2,000	Mallard
Amphibian						
Rocky Mountain Toad	1.84	3.68	25.0	400	2,000	Mallard
Domestic Animals						
Beef Cow	0.522	1.04	3.15	2,000	10,000	Rabbit
Chicken	0.993	1.99	18.7	400	> 2,000	Chicken
Dog	0.251	0.503	5.76	350	1,750	Cat

Table E8-2. Risk Comparison of Estimated Wildlife Doses From Atrazine to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	7.76	77.6	1,040	188	940	Bobwhite
Sage Grouse	0.630	6.30	109	400	>2,000	Pheasant
Bobwhite Quail	2.58	25.8	384	188	940	Bobwhite
American Kestrel	3.04	30.4	457	188	940	Bobwhite
Mammals						
Grasshopper Mouse	10.7	107	1,180	350	1,750	Mouse
Black-tailed Jackrabbit	3.31	33.1	198	134	672	Rat
Pronghorn	0.988	9.88	56.4	134	672	Rat
Coyote	0.317	3.17	48.2	134	672	Rat
Reptiles						
Horned Lizard	1.42	14.2	179	188	940	Bobwhite
Yellow-Bellied Racer	0.372	3.72	66.6	188	940	Bobwhite
Amphibian						
Rocky Mountain Toad	5.82	58.2	873	188	940	Bobwhite
Domestic Animals						
Beef Cow	0.287	2.87	17.8	134	672	Rat
Chicken	0.607	6.07	95.8	188	940	Bobwhite
Dog	0.208	2.08	38.8	134	672	Rat

Table E8-3. Risk Comparison of Estimated Wildlife Doses From Bromacil to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	N/A[a]	62.1	416	350	>1,750	Bobwhite
Sage Grouse	N/A	5.04	43.5	350	>1,750	Bobwhite
Bobwhite Quail	N/A	20.6	154	350	>1,750	Bobwhite
American Kestrel	N/A	24.3	183	350	>1,750	Bobwhite
Mammals						
Grasshopper Mouse	N/A	85.4	472	800	3,998	Rat
Black-tailed Jackrabbit	N/A	26.5	79.3	800	3,998	Rat
Pronghorn	N/A	7.90	22.6	800	3,998	Rat
Coyote	N/A	2.53	19.3	800	3,998	Rat
Reptiles						
Horned Lizard	N/A	11.3	71.8	350	>1,750	Bobwhite
Yellow-Bellied Racer	N/A	2.97	26.6	350	>1,750	Bobwhite
Amphibian						
Rocky Mountain Toad	N/A	46.6	349	350	>1,750	Bobwhite
Domestic Animals						
Beef Cow	N/A	2.29	7.13	800	3,998	Rat
Chicken	N/A	4.85	38.3	350	>1,750	Bobwhite
Dog	N/A	1.67	15.5	800	3,998	Rat

[a]N/A: Not applicable; herbicide not used on rangeland.

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	0.97	1.09	3.64	1,000	>5,000	Bobwhite
Sage Grouse	0.0787	0.0882	0.380	1,000	>5,000	Bobwhite
Bobwhite Quail	0.322	0.361	1.35	1,000	>5,000	Bobwhite
American Kestrel	0.380	0.426	1.60	1,000	>5,000	Bobwhite
Mammals						
Grasshopper Mouse	1.33	1.49	4.13	1,109	5,545	Rat
Black-tailed Jackrabbit	0.414	0.463	0.694	1,109	5,545	Rat
Pronghorn	0.123	0.138	0.198	1,109	5,545	Rat
Coyote	0.0396	0.0443	0.169	1,109	5,545	Rat
Reptiles						
Horned Lizard	0.177	0.198	0.628	1,000	>5,000	Bobwhite
Yellow-Bellied Racer	0.0465	0.0520	0.233	1,000	>5,000	Bobwhite
Amphibian						
Rocky Mountain Toad	0.728	0.815	3.06	1,000	>5,000	Bobwhite
Domestic Animals						
Beef Cow	0.0358	0.0401	0.0624	1,109	5,545	Rat
Chicken	0.0758	0.0849	0.335	1,000	>5,000	Bobwhite
Dog	0.0261	0.0292	0.136	1,109	5,545	Rat

Table E8-5. Risk Comparison of Estimated Wildlife Doses From Clopyralid to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	3.88	93.1	312	293	1,465	Duck
Sage Grouse	0.315	7.56	32.6	293	1,465	Duck
Bobwhite Quail	1.29	30.9	115	293	1,465	Duck
American Kestrel	1.52	36.5	137	293	1,465	Duck
Mammals						
Grasshopper Mouse	5.34	128	354	1,000	>5,000	Mouse
Black-tailed Jackrabbit	1.65	39.7	59.5	860	>4,300	Rat
Pronghorn	0.494	11.9	16.9	860	>4,300	Rat
Coyote	0.158	3.80	14.5	860	>4,300	Rat
Reptiles						
Horned Lizard	0.708	17.0	53.8	293	1,465	Duck
Yellow-Bellied Racer	0.186	4.46	20.0	293	1,465	Duck
Amphibian						
Rocky Mountain Toad	2.91	69.8	262	293	1,465	Duck
Domestic Animals						
Beef Cow	0.143	3.44	5.35	860	>4,300	Rat
Chicken	0.303	7.28	28.7	293	1,465	Duck
Dog	0.104	2.50	11.6	860	>4,300	Rat

Table E8-6. Risk Comparison of Estimated Wildlife Doses From 2,4-D to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	30.5	30.5	102	40	200	Chukar
Sage Grouse	2.30	2.30	9.91	40	200	Chukar
Bobwhite Quail	9.90	9.90	36.8	134	668	Quail
American Kestrel	11.7	11.7	44.1	40	200	Chukar
Mammals						
Grasshopper Mouse	42.0	42.0	116	74	368	Mouse
Black-tailed Jackrabbit	13.0	13.0	18.7	85	424	Rabbit
Pronghorn	3.85	3.85	5.18	80	400	Mule deer
Coyote	1.11	1.11	4.11	20	100	Dog
Reptiles						
Horned Lizard	4.12	4.12	11.5	40	200	Chukar
Yellow-Bellied Racer	0.923	0.923	4.03	40	200	Chukar
Amphibian						
Rocky Mountain Toad	14.6	14.6	53.0	40	200	Chukar
Domestic Animals						
Beef Cow	1.10	1.10	1.56	20	100	Cattle
Chicken	2.23	2.23	8.69	108	541	Chicken
Dog	0.686	0.686	3.19	20	100	Dog

Table E8-7. Risk Comparison of Estimated Wildlife Doses From Dalapon to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	23.3	31.0	572	1,132	5,660	Chicken
Sage Grouse	1.89	2.52	59.8	1,132	5,660	Chicken
Bobwhite Quail	7.73	10.3	211	1,132	5,600	Chicken
American Kestrel	9.12	12.2	252	1,132	5,660	Chicken
Mammals						
Grasshopper Mouse	32.0	42.7	650	920	4,600	Mouse
Black-tailed Jackrabbit	9.92	13.2	109	772	3,860	Rabbit
Pronghorn	2.96	3.95	31.0	1,514	7,570	Rat
Coyote	0.950	1.27	26.5	1,514	7,570	Rat
Reptiles						
Horned Lizard	4.25	5.66	98.7	1,132	5,660	Chicken
Yellow-Bellied Racer	1.12	1.49	36.6	1,132	5,660	Chicken
Amphibian						
Rocky Mountain Toad	17.5	23.3	480	1,132	5,660	Chicken
Domestic Animals						
Beef Cow	0.860	1.15	9.80	772	3,860	Guinea Pig
Chicken	1.82	2.43	52.7	1,132	5,660	Chicken
Dog	0.625	0.834	21.4	772	3,860	Guinea Pig

Table E8-8. Risk Comparison of Estimated Wildlife Doses From Dicamba to Toxicity Reference Levels

| Representative Species | Doses (mg/kg) | | | 1/5 LD$_{50}$ (mg/kg) | LD$_{50}$ (mg/kg) | Reference Species |
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	30.4	30.4	204	135	673	Pheasant
Sage Grouse	2.25	2.25	19.3	135	673	Pheasant
Bobwhite Quail	9.80	9.80	72.9	350	>1,750	Bobwhite
American Kestrel	11.6	11.6	87.3	135	673	Pheasant
Mammals						
Grasshopper Mouse	41.8	41.8	230	238	1,189	Mouse
Black-tailed Jackrabbit	12.9	12.9	36.9	113	566	Rabbit
Pronghorn	3.83	3.83	10.1	151	757	Rat
Coyote	1.08	1.08	7.87	151	757	Rat
Reptiles						
Horned Lizard	3.73	3.73	19.7	135	673	Pheasant
Yellow-Bellied Racer	0.782	0.782	6.73	135	673	Pheasant
Amphibian						
Rocky Mountain Toad	12.4	12.4	88.8	135	673	Pheasant
Domestic Animals						
Beef Cow	1.09	1.09	3.00	151	757	Rat
Chicken	2.18	2.18	16.9	135	673	Pheasant
Dog	0.650	0.650	6.04	151	757	Rat

Table E8-9. Risk Comparison of Estimated Wildlife Doses From Diuron to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	N/A[a]	31.0	832	400	>2,000	Mallard
Sage Grouse	N/A	2.52	86.9	400	>2,000	Mallard
Bobwhite Quail	N/A	10.3	307	400	>2,000	Mallard
American Kestrel	N/A	12.2	366	400	>2,000	Mallard
Mammals						
Grasshopper Mouse	N/A	42.7	945	680	3,400	Rat
Black-tailed Jackrabbit	N/A	13.2	159	680	3,400	Rat
Pronghorn	N/A	3.95	45.2	680	3,400	Rat
Coyote	N/A	1.27	38.6	680	3,400	Rat
Reptiles						
Horned Lizard	N/A	5.66	144	400	>2,000	Mallard
Yellow-Bellied Racer	N/A	1.49	53.3	400	>2,000	Mallard
Amphibian						
Rocky Mountain Toad	N/A	23.3	699	400	>2,000	Mallard
Domestic Animals						
Beef Cow	N/A	1.15	14.3	680	3,400	Rat
Chicken	N/A	2.43	76.6	400	>2,000	Mallard
Dog	N/A	0.834	31.1	680	3,400	Rat

[a]N/A: Not applicable; herbicide not used on rangeland.

Table E8-10. Risk Comparison of Estimated Wildlife Doses From Glyphosate to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	15.5	30.4	103	400	>2,000	Quail
Sage Grouse	1.26	2.46	10.8	400	>2,000	Quail
Bobwhite Quail	5.15	9.47	37.6	400	>2,000	Quail
American Kestrel	6.08	12.1	45.7	400	>2,000	Quail
Mammals						
Grasshopper Mouse	21.4	34.5	110	864	4,320	Rat
Black-tailed Jackrabbit	6.62	1.90	8.50	760	3,800	Rabbit
Pronghorn	1.98	0.451	2.14	864	4,320	Rat
Coyote	0.633	1.01	4.57	864	4,320	Rat
Reptiles						
Horned Lizard	2.83	4.83	17.1	400	>2,000	Quail
Yellow-Bellied Racer	0.744	1.45	6.63	400	>2,000	Quail
Amphibian						
Rocky Mountain Toad	11.6	22.2	86.3	400	>2,000	Quail
Domestic Animals						
Beef Cow	0.573	0.166	0.801	760	3,800	Rabbit
Chicken	1.21	2.03	9.18	400	>2,000	Quail
Dog	0.417	0.832	3.88	760	3,800	Rabbit

Table E8-11. Risk Comparison of Estimated Wildlife Doses From Hexazinone to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	5.20	15.5	281	452	2,258	Bobwhite
Sage Grouse	0.422	1.26	29.3	452	2,258	Bobwhite
Bobwhite Quail	1.73	5.15	104	452	2,258	Bobwhite
American Kestrel	2.04	6.08	123	452	2,258	Bobwhite
Mammals						
Grasshopper Mouse	7.15	21.4	319	338	1,690	Rat
Black-tailed Jackrabbit	2.22	6.62	53.5	172	860	Guinea Pig
Pronghorn	0.662	1.98	15.2	172	860	Guinea Pig
Coyote	0.212	0.633	13.0	172	860	Guinea Pig
Reptiles						
Horned Lizard	0.949	2.83	48.4	452	2,258	Bobwhite
Yellow-Bellied Racer	0.249	0.744	18.0	452	2,258	Bobwhite
Amphibian						
Rocky Mountain Toad	3.90	11.6	236	452	2,258	Bobwhite
Domestic Animals						
Beef Cow	0.192	0.573	4.81	172	860	Guinea Pig
Chicken	0.406	1.21	25.9	452	2,258	Bobwhite
Dog	0.140	0.417	10.5	172	860	Guinea Pig

Table E8-12. Risk Comparison of Estimated Wildlife Doses From Imazapyr to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	7.76	11.6	39.0	430	>2,150	Bobwhite
Sage Grouse	0.630	0.945	4.08	430	>2,150	Bobwhite
Bobwhite Quail	2.58	3.86	14.4	430	>2,150	Bobwhite
American Kestrel	3.04	4.56	17.1	430	>2,150	Bobwhite
Mammals						
Grasshopper Mouse	10.7	16.0	44.3	400	>2,000	Mouse
Black-tailed Jackrabbit	3.31	4.96	7.44	960	>4,800	Rabbit
Pronghorn	0.998	1.48	2.12	400	>2,000	Mouse
Coyote	0.317	0.475	1.81	400	>2,000	Mouse
Reptiles						
Horned Lizard	1.42	2.12	6.73	430	>2,150	Bobwhite
Yellow-Bellied Racer	0.372	0.558	2.50	430	>2,150	Bobwhite
Amphibian						
Rocky Mountain Toad	5.82	8.73	32.8	430	>2,150	Bobwhite
Domestic Animals						
Beef Cow	0.287	0.430	0.668	400	>2,000	Mouse
Chicken	0.607	0.910	3.59	430	>2,150	Bobwhite
Dog	0.208	0.313	1.46	400	>2,000	Mouse

Table E8-13. Risk Comparison of Estimated Wildlife Doses From Mefluidide to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	N/A[a]	1.94	6.50	928	>4,640	Mallard
Sage Grouse	N/A	0.157	0.679	928	>4,640	Mallard
Bobwhite Quail	N/A	0.644	2.40	928	>4,640	Mallard
American Kestrel	N/A	0.760	2.86	928	>4,640	Mallard
Mammals						
Grasshopper Mouse	N/A	2.67	7.38	384	1,920	Mouse
Black-tailed Jackrabbit	N/A	0.827	1.24	384	1,920	Mouse
Pronghorn	N/A	0.247	0.353	384	1,920	Mouse
Coyote	N/A	0.0792	0.302	384	1,920	Mouse
Reptiles						
Horned Lizard	N/A	0.354	1.12	928	>4,640	Mallard
Yellow-Bellied Racer	N/A	0.0929	0.416	928	>4,640	Mallard
Amphibian						
Rocky Mountain Toad	N/A	1.46	5.46	928	>4,640	Mallard
Domestic Animals						
Beef Cow	N/A	0.0717	0.111	384	1,920	Mouse
Chicken	N/A	0.152	0.599	928	>4,640	Mallard
Dog	N/A	0.0521	0.243	384	1,920	Mouse

[a]N/A: Not applicable; herbicide not used on rangeland.

Table E8-14. Risk Comparison of Estimated Wildlife Doses From Metsulfuron Methyl to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	N/A[a]	0.582	1.95	502	>2,510	Mallard
Sage Grouse	N/A	0.0472	0.204	502	>2,510	Mallard
Bobwhite Quail	N/A	0.193	0.721	502	>2,510	Mallard
American Kestrel	N/A	0.228	0.857	502	>2,510	Mallard
Mammals						
Grasshopper Mouse	N/A	0.801	2.21	1,000	>5,000	Rat
Black-tailed Jackrabbit	N/A	0.248	0.372	1,000	5,000	Rat
Pronghorn	N/A	0.0741	0.106	1,000	>5,000	Rat
Coyote	N/A	0.0238	0.0905	1,000	>5,000	Rat
Reptiles						
Horned Lizard	N/A	0.106	0.336	502	>2,510	Mallard
Yellow-Bellied Racer	N/A	0.0279	0.125	502	>2,510	Mallard
Amphibian						
Rocky Mountain Toad	N/A	0.437	1.64	502	>2,510	Mallard
Domestic Animals						
Beef Cow	N/A	0.0215	0.0334	1,000	>5,000	Rat
Chicken	N/A	0.0455	0.180	502	>2,510	Mallard
Dog	N/A	0.0156	0.0728	1,000	>5,000	Rat

[a]N/A: Not applicable; herbicide not used on rangeland.

Table E8-15. Risk Comparison of Estimated Wildlife Doses From Picloram to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD₅₀ (mg/kg)	LD₅₀ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme	$1/5\ LD_{50}$ (mg/kg)	LD_{50} (mg/kg)	
Birds						
Lark Bunting	14.9	22.3	74.7	400	>2,000	Pheasant
Sage Grouse	0.992	1.49	6.35	400	>2,000	Pheasant
Bobwhite Quail	4.65	6.98	25.9	400	>2,000	Pheasant
American Kestrel	5.55	8.32	31.2	400	>2,000	Pheasant
Mammals						
Grasshopper Mouse	20.4	30.6	84.0	400	2,000	Mouse
Black-tailed Jackrabbit	6.31	9.47	12.8	400	2,000	Rabbit
Pronghorn	1.85	2.78	3.36	144	>720	Sheep
Coyote	0.443	0.664	2.28	400	2,000	Mouse
Reptiles						
Horned Lizard	0.903	1.36	1.36	400	>2,000	Pheasant
Yellow-Bellied Racer	0.0389	0.0583	0.0602	400	>2,000	Pheasant
Amphibian						
Rocky Mountain Toad	0.755	1.13	1.16	400	>2,000	Pheasant
Domestic Animals						
Beef Cow	0.516	0.774	0.913	144	>720	Sheep
Chicken	0.968	1.45	5.51	400	>2,000	Pheasant
Dog	0.233	0.349	1.62	400	2,000	Mouse

Table E8-16. Risk Comparison of Estimated Wildlife Doses From Simazine to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	31.0	31.0	1,040	928	>4,640	Mallard
Sage Grouse	2.52	2.52	109	928	>4,640	Mallard
Bobwhite Quail	10.3	10.3	384	928	>4,640	Mallard
American Kestrel	12.2	12.2	457	928	>4,640	Mallard
Mammals						
Grasshopper Mouse	42.7	42.7	1,180	1,000	>5,000	Mouse
Black-tailed Jackrabbit	13.2	13.2	198	1,000	>5,000	Rabbit
Pronghorn	3.95	3.95	56.4	1,000	>5,000	Mouse
Coyote	1.27	1.27	48.2	1,000	>5,000	Mouse
Reptiles						
Horned Lizard	5.66	5.66	179	928	>4,640	Mallard
Yellow-Bellied Racer	1.49	1.49	66.6	928	>4,640	Mallard
Amphibian						
Rocky Mountain Toad	23.3	23.3	873	928	>4,640	Mallard
Domestic Animals						
Beef Cow	1.15	1.15	17.8	1,000	>5,000	Mouse
Chicken	2.43	2.43	95.8	928	>4,640	Mallard
Dog	0.834	0.834	38.8	1,000	>5,000	Mouse

Table E8-17. Risk Comparison of Estimated Wildlife Doses From Sulfometuron Methyl to Toxicity Reference Levels

| Representative Species | Doses (mg/kg) | | | 1/5 LD$_{50}$ (mg/kg) | LD$_{50}$ (mg/kg) | Reference Species |
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	N/A[a]	4.34	14.6	1,000	>5,000	Mallard
Sage Grouse	N/A	0.353	1.52	1,000	>5,000	Mallard
Bobwhite Quail	N/A	1.44	5.38	1,000	>5,000	Mallard
American Kestrel	N/A	1.70	6.40	1,000	>5,000	Mallard
Mammals						
Grasshopper Mouse	N/A	5.98	16.5	1,000	>5,000	Rat
Black-tailed Jackrabbit	N/A	1.85	2.78	1,000	>5,000	Rat
Pronghorn	N/A	0.553	0.790	1,000	>5,000	Rat
Coyote	N/A	0.177	0.675	1,000	>5,000	Rat
Reptiles						
Horned Lizard	N/A	0.793	2.51	1,000	>5,000	Mallard
Yellow-Bellied Racer	N/A	0.208	0.932	1,000	>5,000	Mallard
Amphibian						
Rocky Mountain Toad	N/A	3.26	12.2	1,000	>5,000	Mallard
Domestic Animals						
Beef Cow	N/A	0.161	0.249	1,000	>5,000	Rat
Chicken	N/A	0.340	1.34	1,000	>5,000	Mallard
Dog	N/A	0.117	0.544	1,000	>5,000	Rat

[a]N/A: Not applicable; herbicide not used on rangeland.

Table E8-18. Risk Comparison of Estimated Wildlife Doses From Tebuthiuron to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	3.88	11.6	416	400	>2,000	Bobwhite
Sage Grouse	0.315	0.945	43.5	400	>2,000	Bobwhite
Bobwhite Quail	1.29	3.86	154	400	>2,000	Bobwhite
American Kestrel	1.52	4.56	183	400	>2,000	Bobwhite
Mammals						
Grasshopper Mouse	5.34	16.0	472	106	>528	Mouse
Black-tailed Jackrabbit	1.65	4.96	79.3	57	286	Rabbit
Pronghorn	0.494	1.48	22.6	106	>528	Mouse
Coyote	0.158	0.475	19.3	100	>500	Dog
Reptiles						
Horned Lizard	0.708	2.12	71.8	400	>2,000	Bobwhite
Yellow-Bellied Racer	0.186	0.558	26.6	400	>2,000	Bobwhite
Amphibian						
Rocky Mountain Toad	2.91	8.73	349	400	>2,000	Bobwhite
Domestic Animals						
Beef Cow	0.143	0.430	7.13	106	>528	Mouse
Chicken	0.303	0.910	38.3	100	>500	Chicken
Dog	0.104	0.313	15.5	100	>500	Dog

Table E8-19. Risk Comparison of Estimated Wildlife Doses From Triclopyr to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	11.6	30.4	207	340	1698	Mallard
Sage Grouse	0.945	2.46	21.6	340	1698	Mallard
Bobwhite Quail	3.86	9.47	75.2	340	1698	Mallard
American Kestrel	4.56	12.1	91.4	340	1698	Mallard
Mammals						
Grasshopper Mouse	16.0	34.5	220	94	471	Mouse
Black-tailed Jackrabbit	4.96	1.90	17.0	110	550	Rabbit
Pronghorn	1.48	0.451	4.29	62	310	Guinea Pig
Coyote	0.475	1.01	9.14	62	310	Guinea Pig
Reptiles						
Horned Lizard	2.12	4.83	34.2	340	1698	Mallard
Yellow-Bellied Racer	0.558	1.45	13.3	340	1698	Mallard
Amphibian						
Rocky Mountain Toad	8.73	22.2	173	340	1698	Mallard
Domestic Animals						
Beef Cow	0.430	0.166	1.60	62	310	Guinea Pig
Chicken	0.910	2.03	18.4	340	1698	Mallard
Dog	0.313	0.832	7.76	62	310	Guinea Pig

Table E8-20. Risk Comparison of Estimated Wildlife Doses From Diesel Oil to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	16.5	16.5	55.3	3,280	>16,400	Mallard
Sage Grouse	1.66	1.66	7.24	3,280	>16,400	Mallard
Bobwhite Quail	5.89	5.89	22.2	3,280	>16,400	Mallard
American Kestrel	6.88	6.88	26.0	3,280	>16,400	Mallard
Mammals						
Grasshopper Mouse	22.8	22.8	63.6	1,476	>7,380	Rat
Black-tailed Jackrabbit	7.07	7.07	12.0	1,476	>7,380	Rat
Pronghorn	2.16	2.16	3.70	1,476	>7,380	Rat
Coyote	0.920	0.920	3.75	1,476	>7,380	Rat
Reptiles						
Horned Lizard	5.73	5.73	21.1	3,280	>16,400	Mallard
Yellow-Bellied Racer	1.80	1.80	8.26	3,280	>16,400	Mallard
Amphibian						
Rocky Mountain Toad	28.0	28.0	108	3,280	>16,400	Mallard
Domestic Animals						
Beef Cow	0.660	0.660	1.31	1,476	>7,380	Rat
Chicken	1.58	1.58	6.46	3,280	>16,400	Mallard
Dog	0.693	0.693	3.24	1,476	>7,380	Rat

Table E8-21. Risk Comparison of Estimated Wildlife Doses From Kerosene to Toxicity Reference Levels

Representative Species	Doses (mg/kg)			1/5 LD$_{50}$ (mg/kg)	LD$_{50}$ (mg/kg)	Reference Species
	Rangeland Typical	Right-of-Way Typical	Right-of-Way Extreme			
Birds						
Lark Bunting	16.5	16.5	55.3	3,280	>16,400	Mallard
Sage Grouse	1.66	1.66	7.24	3,280	>16,400	Mallard
Bobwhite Quail	5.89	5.89	22.2	3,280	>16,400	Mallard
American Kestrel	6.88	6.88	26.0	3,280	>16,400	Mallard
Mammals						
Grasshopper Mouse	22.8	22.8	63.6	5,600	>28,000	Rat
Black-tailed Jackrabbit	7.07	7.07	12.0	5,600	>28,000	Rat
Pronghorn	2.16	2.16	3.70	5,600	>28,000	Rat
Coyote	0.920	0.920	3.75	5,600	>28,000	Rat
Reptiles						
Horned Lizard	5.73	5.73	21.1	3,280	>16,400	Mallard
Yellow-Bellied Racer	1.80	1.80	8.26	3,280	>16,400	Mallard
Amphibian						
Rocky Mountain Toad	28.0	28.0	108	3,280	>16,400	Mallard
Domestic Animals						
Beef Cow	0.660	0.660	1.31	5,600	>28,000	Rat
Chicken	1.58	1.58	6.46	3,280	>16,400	Mallard
Dog	0.693	0.693	3.24	5,600	>28,000	Rat

Table E8-22. Risks to Aquatic Species

Herbicide	Rangeland Drift on Pond, Typical	Rights-of-Way Drift on Pond, Typical	Accidental Direct Spray of Pond	Helicopter Jettison into Pond	Trout	Bluegill	Fathead Minnow	Aquatic Inverte-brate[e]
	Estimated Environmental Concentrations (mg/L)				**Toxicity Values (mg/L) 1/10 LC$_{50}$ (1/2 LC$_{50}$)**			
Amitrole	0.00328	0.00328	0.910	11.8	325 (1,625)[b]	120 (600)	10 (50)	0.143 (0.715)
Atrazine	0.00164	0.00656	3.68	11.8	2.4 (12)	4.2 (21)	1.5 (7.5)	0.115 (0.575)
Bromacil	—[e]	0.0131	1.47	5.88	7.5 (37.5)	7.1 (35.5)	NA	4.0 (20)
Chlorsulfuron	—[e]	0.000231	0.0129	58.8	25 (125)	25 (125)	NA	NA
Clopyralid	0.00082	0.0197	1.10	58.8	10.4 (52)	12.5 (62.5)	NA	0.1 (0.5)
2,4-D	0.00656	0.00656	0.736	11.8	0.9 (4.5)	0.12 (0.6)	0.33 (1.65)	0.56 (2.8)
Dalapon	0.00492	0.00656	2.02	4.41	34 (170)	10.5 (52.5)	29 (145)	0.6 (3.0)
Dicamba	0.00656	0.00656	0.736	11.8	2.8 (14)	5.0 (25)	NA	1.1 (5.5)
Diuron	—[e]	0.00656	2.94	9.41	0.49 (2.45)	0.82 (4.1)	NA	0.14 (0.7)
Glyphosate	0.00656	0.00656	0.460	14.7	3.8 (19)	2.4 (12)	9.7 (48.5)	78 (390)
Hexazinone	0.00736	0.00328	0.993	8.82	32 (160)	37 (185)	20.7 (103.5)	12.5 (62.5)
Imazapyr	0.00164	0.00246	0.138	58.8	10 (50)	10 (50)	NA	10 (50)
Mefluidide	—[e]	0.000410	0.0230	58.8	10 (50)	10 (50)	NA	NA
Metsulfuron methyl	—[e]	0.000123	0.00689	58.8	15 (75)	15 (75)	NA	15 (75)
Picloram	0.00328	0.00492	0.276	8.82	1.25 (6.25)	2.3 (11.5)	NA	38 (190)
Simazine	—[e]	0.00656	3.68	17.6	0.28 (1.4)	1.16 (5.8)	0.35 (1.75)	0.056 (0.28)
Sulfometuron methyl	—[e]	0.000923	0.0507	58.8	1.25 (6.25)	1.25 (6.25)	0.12 (0.60)	1.25 (6.25)
Tebuthiuron	0.00082	0.00246	1.47	17.6	14.4 (61)	11.2 (56)	16 (80)	29.7 (148.5)
Triclopyr	0.00246	0.00656	0.735	23.5	11.7 (58.5)	14.8 (74)	8.85 (44.25)	103 (515)
Diesel oil	0.00328	0.00328	0.184	14.7	0.019 (0.095)[c]	0.081 (0.405)[b]	0.69 (3.45)[d]	0.021 (0.105)
Kerosene	0.00328	0.00328	0.184	14.7	0.003 (0.015)	NA	NA	NA

N/A: Not available.
[a]Values are for *Daphnia* sp., except diesel oil (value for pink shrimp used).
[b]Value is for salmon.
[c]Value is for unspecified freshwater fish.
[d]Value is for sheepshead minnow.
[e]Not applied to rangeland in this program.

Appendix F
Fire Ecology of Western Plant Species

Principles and Processes of Fire Effects

Introduction

Fires that burn under similar weather conditions often have a surprisingly wide range of effects on vegetation. Plant communities do not respond in one specific way to fire. Most fire effects are related to factors other than the heat released during the active flaming passage of the fire front. Any given site with its fuel complex and plant community can burn in a variety of ways, and consequently a wide range of fire effects can result.

The most significant sources of heat from most fires are downed dead surface fuels, litter, and duff layers. However, dead branches, leaves, or needles on standing plants can produce a considerable amount of heat. Old, fairly decadent stands of shrubs, for example, may produce a much more intense fire than a young shrub stand, which may have so little dead or dry material that it cannot be ignited. The amount of dead woody fuel, thickness of litter and duff layers, and amount of dead material on a living plant may be significantly greater than would normally occur under "natural conditions" if fire has been excluded from an environment in which fires used to occur at a fairly high frequency. In this situation, fire-caused mortality may be much higher than it would have been under natural conditions because of the greater potential for production of lethal temperatures. Moisture content is the most significant factor influencing the proportion of material in each fuel category that will burn.

Fireline intensity is directly related to the length of the flames and is a measure of the rate of heat released by the flames. Most of this heat is directed upward. Burn or fire severity is a qualitative measure of the amount of heat that is directed downward, which is available to heat soils and belowground plant parts. A low severity fire removes little surface fuel or organic material and causes little subsurface heating. A moderate severity fire removes some of the fuel in small and medium size classes, may consume some of the forest floor organic layer (or litter and

organic layer beneath rangeland shrubs), and can heat surface soil layers. A high severity fire consumes most of the organic material on the soil surface and can cause significant amounts of subsurface heating.

Subsurface heating and associated mortality depends on the amount of surface dead fuel, the amount and compactness of litter and duff, and the moisture content of those materials, all of which affect fire duration—the length of time a heat source is present (Wade 1986). Soil moisture also retards penetration of heat into soil layers (Shearer 1975, Frandsen and Ryan 1986). If moderate to heavy accumulations of surface dead fuel or organic layers exist, their consumption in smoldering and glowing combustion are the best indicators of significant amounts of subsurface heating and potential damage to buried plant parts.

Different plant species have specific physical characteristics that determine their sensitivity to fire heating. Given the fuel characteristics and moisture conditions on a particular site when it is burned, the fire creates a given amount of heat that lasts for a specific amount of time and causes a certain degree of injury to a plant. Different plant species have different methods for recovery after fire. A plant's ability to recover can be affected by factors that vary with the growing season or with the age of the plant. Whether the plants that first appear after a fire can successfully establish on the site can be significantly affected by such external factors as postfire weather, postfire animal use, and plant competition.

Plant Mortality

Plant mortality depends on whether tissue is lethally heated. The lowest temperature at which plant cells are killed is approximately 50 to 55 °C (122 to 131 °F) (Baker 1929, as cited in Wright and Bailey 1982). Actual death depends on the temperature reached and the duration of time to which plant tissue is exposed to that temperature. Additionally, some plant tissues, particularly growing points (meristems or buds), tend to be much more sensitive to fire heat when they are actively growing and their tissue moisture is high than when tissue moisture content is low (Wright

and Bailey 1982). Thus, plant tissues die more readily after exposure to a specific temperature for a certain length of time when actively growing than when they are physiologically dormant or quiescent, or have finished active growth for the year.

Structural and physical characteristics affect whether the aboveground part of a woody plant will be killed by a fire. Important crown characteristics include branch density, location of the base of the crown with respect to surface fuels, total crown size (Brown and Davis 1973), and bud size, with small size buds more likely to be lethally heated than large buds, or buds covered by long needles (Wagener 1961, as cited in Ryan 1982). The moisture content of the foliage also affects its flammability. Moisture content of new leaves and twigs varies throughout the growing season, being highest when active growth is occurring. These tissues also tend to be more sensitive to heating when actively growing because of their high moisture content. The moisture content of old conifer foliage is lowest in the spring and generally increases to a maximum level in late summer (Norum and Miller 1984, Chrosowiecz 1986). The moisture content of understory plants can be high enough to prevent fire from entering the plant community. Evergreen plants are usually more flammable than deciduous plants, because their moisture content tends to be lower and because evergreens often store flammable compounds in their leaves. The mortality of tree and shrub crowns is directly related to the amount of heating the crown receives from the surface fire, and the crown is always killed if flames burn through the entire crown. The amount of crown scorch can be calculated from flame length, ambient air temperature, and windspeed (Albini 1976).

Trees and shrubs also can be killed by lethally heating the cambium, the active growth layer that lies beneath the bark. Bark surface texture can affect its likelihood of ignition, whether stringy and flammable, or smooth. The fire resistance of tree stems is most closely related to bark thickness, which varies by species and with age. The cambium layer of thin bark trees, such as lodgepole pine, subalpine fir, aspen, madrone, or most of the spruces, is usually dead beneath any charred bark. The cambium beneath thick bark layers, such as those present on ponderosa pine, Douglas-fir, and western larch, is usually only killed by heat released over a long duration, such as from burnout of logs, and deep litter and duff layers. The cambium of shrubs is usually killed by fire because shrub stems tend

to be small in diameter and have relatively thin bark.

Root mortality can kill plants even if the aboveground parts of the plants receive little damage. The likelihood of damage to roots depends on their location with respect to the surface of the ground; whether they are located in flammable organic layers or in soil; how close they are to accumulations of dead woody fuels that may burn for a long time and produce enough heat to damage them; and the moisture content of the fuel, organic layers, and soil. Structural support roots that grow laterally near the surface are more susceptible to fire damage and consumption than roots that grow downward. Roots typically found in organic layers are more likely to be lethally heated or consumed than those located in soil layers, and trees mostly rooted in organic matter are more subject to postfire windthrow. Tree feeder roots, those that collect most of its water and nutrients, are very small in diameter and usually grow laterally near the surface. If a large proportion of the feeder roots are located in soil organic layers rather than in soil, they are much more subject to lethal heating. Root mortality is closely related to burn severity, the amount of consumption of large-diameter dead woody fuels, the reduction in depth of organic layers caused by fire, and the related amount of soil heating.

Postfire Sprouting

Postfire sprouting is a means by which many plants recover after fire. The same factors that control mortality of roots affect mortality of reproductive structures of woody plants, grasses, and forbs. The reproductive parts of plants are located at various levels in the litter, duff, and soil layers, and thus have differing susceptibility to heat generated at the surface. The type of medium in which these parts grow and the depth below the surface at which dormant buds occur may be a species-specific characteristic (Flinn and Wein 1977).

Many plants have buds located in the tissue of upright stems, above or below the surface of the ground. Buds may be located on laterally growing stems, such as stolons or rhizomes, which for different species are characteristically located at different depths below the surface of the ground. Some trees have root collar buds located in stem tissue at the point where roots spread out from the base of the stem. Lignotubers, burls, and root crowns are all names for masses of woody tissue from which roots and stems originate and which are often covered with dormant buds (James 1984). Dormant buds may be deeply buried in wood

and may be located far below the surface if the tissue mass is large. Forbs often regenerate from buried stemlike structures or from bulbs or corms. Some species have dormant buds or bud primordia located along the surface of roots from which new shoots can originate.

Most of these dormant buds are prevented from growing by compounds that are translocated from aboveground parts of the plants to buds on roots and rhizomes. Fire removes the source of these inhibitors when it kills the top of the plants. Compounds in roots then promote bud outgrowth (Schier et al. 1985). Postfire sprouting ability can vary with plant age. Young plants that developed from a seed may not be able to sprout until they reach a certain age, while some older plants gradually lose their ability to sprout.

A key factor in determining whether a plant is capable of producing new shoots after a fire depends on whether dormant buds were lethally heated by the fire. Plant parts deep below the soil surface, in moist soil or organic layers, or in areas where there is little surface fuel to provide a heat source are most likely to survive. For forest and for rangeland situations, postfire sprouting can be closely linked to the severity of the burn with respect to the location of reproductive structures with dormant buds (Dyrness and Norum 1983, Morgan and Neuenschwander 1988). The number of postfire sprouts can be related to depth of burn and the amount of stimulation or death of dormant buds.

Many species of grass are rhizomatous, with meristems and buds buried beneath the litter, duff, or soil surface. Whether rhizomatous grasses are stimulated or killed by fire depends on rhizome location with respect to the surface; whether rhizomes are located in soil or organic layers; the moisture content of the litter, organic, and soil layers; and the amount and duration of heat generated by the surface fire. Rhizomatous grasses often respond positively to rangeland fires because meristems and buds are usually protected by soil; in forested areas, they are more likely to be damaged because there is more surface fuel, which can cause more subsurface heating lethal to rhizomes.

Many of the grasses in the EIS region are bunchgrasses, which tend to be more sensitive to fire than rhizomatous grasses. The sensitivity of bunchgrasses to fire depends on several factors, including the relative location of growing points and buds with respect to the soil surface, the amount of fuel within the bunch (which relates to bunchgrass diameter), and bunchgrass coarseness (Wright 1971). Fire tends to pass fairly quickly through coarse-textured bunchgrasses as compared to fine-textured species. Fine-leaved bunchgrasses often have a concentration of dense plant material at their bases, and therefore have the capacity to concentrate more heat at their growing points if basal fuels ignite when dry and then smolder (Wright and Bailey 1982).

Actively growing bunchgrasses are more sensitive to fire than dormant bunchgrasses (Wright 1970). However, these sagebrush/grass communities rarely burn during this actively growing stage because grasses and shrubs have high foliage moisture. The exception is sites where bunchgrasses are growing in association with annual grasses that cure out and become flammable while perennial grasses are still physiologically active. Prescribed fires would not be staged during this time because of potential harm to perennial plants.

Reproduction From Seed

Many plant species reproduce from seed. Seedbed requirements vary among species; some seedlings do well on organic seedbeds, but most do better on bare mineral soil, particularly in drier climates or climates with summer dry periods. Seeds may come from onsite unburned tree canopies, fall from cones that opened because of the heat of the fire, be blown or carried onto the site by animals, or have been stored in organic or soil layers. Seed caches of rodents and birds can provide an important postfire seed source (West 1968, Tomback 1986). Some species of plants, such as ceanothus, can store seed that remains viable for 200 to 300 years (Gratkowski 1962, as cited in Noste and Bushey 1987). Some of these buried seeds are stimulated to germinate by heat or by materials washed from charred wood left after the fire (Keeley 1987). Fire can also improve the variety of seeds that germinate by removing the litter layers beneath plants such as juniper and pinyon. These litter layers contain chemicals that inhibit germination of seeds of other species (Everett 1987a). The age at which plants begin to produce seed and whether seed production declines significantly for a particular species as the plant ages also affect postfire seed reproduction.

Effect of Season of Burning

The season in which a burn occurs can result in different fire effects for several reasons. The season of a prescribed fire determines whether a plant is actively growing (and more susceptible to fire heating) or is dormant (with tissue more resistant to the effects of heat). However, the exact timing of plant growth can differ considerably among years, depending on temperature, precipitation, and snowpack melting (Sauer and Uresk 1976, Turner and Randall 1987). Prescribed fires done in the spring or fall take into consideration the state of growth of the plant, as well as the site moisture regime typical for that time of year. The onset of growth and timing of dormancy for a particular species will vary for a particular year or with the elevation, aspect, and geographic location of the plant community.

The levels of starches and sugars, or carbohydrates, that plants use for energy and growth are subject to a seasonal cycle. Levels of stored carbohydrate decline early in the growing season and whenever requirements for growth of the plant exceed the production of carbohydrates by photosynthesis (Cook 1966a and 1966b). Initial new growth of sprouts and suckers relies on stored starches and sugars. If a fire occurs when stored carbohydrates are low, or at a time in the growing season when a plant does not have time to manufacture enough carbohydrates to survive a subsequent period of drought or cold (Mueggler 1983), a plant may die, or its production in the subsequent year(s) may be much reduced (Trlica 1977).

Effect of Weather

Prefire weather determines the growth stage of the plant, the amount of production in the present year and previous years and thus the amount of herbaceous fuel, and the vigor of the plants. A drought-stressed plant is less likely to survive a fire, and, if it lives, its production in the next few years may be greatly reduced compared to a plant in good condition when it is burned. Prefire weather also determines the moisture regime on the site—whether fuels in different size classes, organic layers, and soil are wet, moist, dry, or very dry. Postfire weather, particularly temperature and precipitation, can determine whether seedlings successfully establish, whether plants produce sprouts that fall or wait until the following spring, whether sprouts survive a subsequent period of drought or cold, and the rate of recovery of the plant

community, especially the amount of postfire production of plants.

Postfire Plant Productivity

Fire can have a significant effect on postfire plant productivity. There may be a significant decrease during the initial postfire recovery period, then an increase in production after 1 or several years. Productivity may increase after the first growing season. Total productivity may not change significantly, but can shift among classes of plants on the site, such as from conifers that are killed by a fire to shrubs, grasses, and forbs. Total vegetative productivity may actually decrease, but shift from less desirable to more desirable species, as from woody plants to grasses and forbs. Immediate productivity increases are usually more likely if significant amounts of vegetative reproduction or regeneration occur than if the site must reestablish from seed. Sprouts can obtain nutrients and carbohydrates for initial growth from the parent plant, while a seedling often has access to only a small nutrient reserve in seed and may grow fairly slowly initially. Seedling establishment and growth depend much more on site conditions.

Greatly increased amounts of flowering and fruiting may occur, including a significantly enhanced output of grass seed and berries (Daubenmire 1975, Christensen and Mueller 1975, Young 1986). The same factors that increase vegetative productivity—warmer temperatures, improved nutrient availability, and removal of senescent, woody material that requires a lot of energy to maintain—cause changes in production. The amount of flowering and fruiting may decrease over prefire levels for some time if plants were severely damaged by burning. Whether increases or decreases in production of specific species are desirable and are achieved by a prescribed fire depends on the specific objectives for burning.

Relationship of Fire to Animal Use

If burning occurs in close association with heavy use of the plant community by livestock or wildlife, either before or after the burn, plant recovery may be delayed or prevented. Heavy prefire use may deplete plant carbohydrate reserves. Heavy postfire use of perennial plants in the first growing season after a fire is likely to cause the most harm, particularly in arid and semi-arid range communities (Trlica 1977). Livestock and wildlife are often attracted to burned areas because of increased palatability, availability, and the earlier spring

greenup that often occurs on burned rangelands and grasslands.

Depending on the plant community and its production capabilities, some use after the first full growing season may not have a negative impact and indeed may be desirable, as in tobosagrass communities. In most cases, however, two full growing seasons of postfire rest are necessary before plants can sustain much use (Wright and Bailey 1982). A longer recovery period is necessary if weather has been unfavorable for growth or if establishment of plants from seeds is required to completely revegetate the site. Desert plants required more than 7 years of recovery after moderate defoliation (Cook and Child 1971 in Trlica 1977), and some shrubland sites may require such a long period of postfire rest if recovery of browse species is desired.

Postfire Plant Competition

Plants compete for whatever is in shortest supply—particularly water, nutrients, and light. Whether competition occurs and the degree to which it occurs depend on the species present on the site, the number of plants present, and the site conditions. Species characteristics may dictate that plants need the same things from the environment at the same time, such as the timing of germination and growth, germination and establishment requirements, rate of growth, and requirements for water and nutrients.

Fire has a significant effect on plant competition by changing the numbers and species of existing plants, altering site conditions, and inducing a situation where many plants must reestablish on a site. In a postfire situation, established perennial plants that are recovering vegetatively usually have an advantage over plants that are developing from seed, because they can take up water and nutrients from an existing root system while seedlings must develop a new root system. Sprouting plants may rapidly develop a crown which can shade out or limit growth of other plants. Natural regeneration of shrubs may severely limit growth of naturally occurring or planted conifers because of competition for light or moisture (Stein 1986). Grass seeded for postfire erosion control in forested areas may easily overtop conifer seedlings (Miller 1988) and in chaparral areas, may compete with sprouts and seedlings of native plants (Barro and Conard 1987). Litter from seeded grasses may also increase the flammability of the site to much higher levels than would occur if native vegetation only recovered on the site (Cohen 1986, as cited in Barro and

Conard 1987). A second fire after a short term interval might kill all seedlings of native species, often before they have produced much seed. Numbers and vigor of native plants would be further reduced. Cheatgrass seedlings can grow roots at much cooler soil temperatures than many native perennial grass seedlings, and use up soil moisture in the spring before other species get their roots down into the soil profile (Thill, Beck, and Callihan 1984).

A lack of fire can also increase plant competition. For example, junipers can take up increasing amounts of soil water in sagebrush/grass communities into which they have invaded, and eventually exclude most other species because of moisture limitations. Grass production tends to decrease as sagebrush cover increases, again because of competition for water. Young stands of conifers which develop in the absence of fire beneath mature overstories of ponderosa pine compete for moisture and nutrients with the mature trees, weakening them and making them susceptible to insects and disease. Removal of these competing plants with fire, in accordance with management objectives for the site, is the most efficient way to manage these plant communities. Management objectives for the site often require the removal of these competing plants. Depending on the site, prescribed fire, or fire in combination with other treatments, is the most efficient and ecologically sound way to manage these plant communities.

Control of Fire Effects With a Prescribed Fire Prescription

Prescribed fire prescriptions and the prescribed fire plan are developed with regard to site characteristics and the reproductive characteristics of the plant species present on the site. Fire effects on a particular plant community or species can thus be controlled through the choice of weather and fuel moisture conditions under which the fire is staged, the time of the year when the site is burned, the size of the burned area as it relates to postfire livestock and wildlife use, and pre- and postfire site management. If livestock use of a prescribed fire site can not be properly managed the site should not be burned. For some plant communities in poor condition or dominated by undesired species, it may be necessary to artificially reseed the area after burning because natural revegetation by desired species is unlikely to occur.

If undesired species are present on a site that may respond positively to prescribed fire, it may be possible to choose a prescription for burning that will favor other species. In some situations, a better choice may be to not burn that site and to select another treatment that optimizes positive effects.

Much of the literature about fire effects is based on observations of plant response after wildfires, which generally occur under burning conditions much more severe than those for a prescribed fire. In some instances, tradeoffs are made with prescribed burning; some short-term undesirable effects on preferred species have to occur to have desired results on species that are targets for removal. The following literature review about the effects of fire on species in the analysis regions within the EIS area describes the types of responses that the key plant species can make and explains why these responses occur. Some inferences about prescribed fire effects are derived from the study of wildfires because detailed prescribed fire studies are few.

Fire Effects by Vegetation Analysis Region

Sagebrush Analysis Region

Shrubs

Big sagebrush is almost always killed by fire, and because it does not sprout, it may be decades before it establishes preburn cover (Harniss and Murray 1973). The rate of reestablishment depends on the size of the area burned, postfire grazing management practices, and the subspecies of sagebrush. Seed germination of mountain big sagebrush is enhanced by low and moderate severity fires (Hironaka et al. 1983). Seeds of basin and Wyoming big sagebrush are not known to be heat stimulated. Recolonization of sites by these species generally takes longer than in a mountain big sagebrush site. Black sagebrush and low sagebrush are both killed by fire, and must also develop on a burned area from seed carried from off the site (McMurray 1987c, Tirmenstein 1986a). Historically, fire has been a rare occurrence in black sagebrush and low sagebrush communities because there are few associated herbaceous plants to carry the fire.

Other important species of the sagebrush analysis region can reproduce by sprouting after a fire. Both rubber (gray) rabbitbrush (Bradley 1986a) and low (green) rabbitbrush (Bradley 1986b) sprout after fires from buds located at or just below the soil surface,

although rubber rabbitbrush is considered to be somewhat more fire sensitive than low rabbitbrush. Plants that survive a fire can produce copious quantities of seed if there are few surviving perennial grasses and forbs to provide competition. Sprouting rabbitbrush plants and newly established seedlings can dominate rabbitbrush sites in poor condition. However, these plants are generally short-lived, and sagebrush will usually gain dominance over rabbitbrush. A few rabbitbrush plants usually do survive in old sagebrush stands and may gain short-term importance after the next fire or other major disturbance.

The two species of horsebrush most commonly found in this analysis region, gray horsebrush and littleleaf horsebrush, burn fairly rapidly during a fire and transfer little heat below the surface, although more heat will be transferred under dry soil conditions. They will sprout vigorously from any root crowns that are not damaged by the fire, may establish many new plants from seedlings, and may gain dominance over a sagebrush site until sagebrush plants increase in number and size (Ahlenslager 1986b, Ahlenslager 1986c).

Little information is available about the response of desert snowberry to fire. It has been reported to resprout after a fire in Nevada (Klebenow and Beall 1977, as cited in McMurray 1986d). It is similar in form to mountain snowberry, which can resprout from root crowns and rhizomes and readily develops from seed. As with other shrubs, its ability to sprout relates to the location of its buds and the severity of the fire.

Bitterbrush is a species of special interest because it has such valuable forage and browse qualities. Bitterbrush has two major growth forms, one that is decumbent, with branches touching the ground, and the other form taller and more erect. The decumbent form is more likely to resprout after a fire (Nord 1965, as cited in Bradley 1986f). The erect form, which is more likely to be found in sagebrush communities, has dormant buds located in stem tissue at or near the surface and tends to be fairly fire sensitive. Older plants seem to produce fewer sprouts than younger plants (Bradley 1986f). Both forms reproduce after fires from rodent-cached seed. Because bitterbrush plants die of old age, fire seems to be necessary for maintenance of the species, even though mortality of plants during any fire may be fairly high. Mortality is minimized by burning when soils are moist, either in the spring or late in the fall after plants have become dormant and rain has

fallen. Bitterbrush mortality is highest when fuel consumption is high.

Grasses

Many of the dominant grass species of the sagebrush analysis region are fairly fire resistant and can produce new shoot growth even after moderate to high severity burns. Bluebunch wheatgrass regenerates by sprouting after a fire, as little fuel is concentrated at the base of the plant and basal growing points are generally not damaged (Antos et al., as cited in Bradley 1986e). Sandberg bluegrass also is fairly resistant to fire damage because of its small bunch size, and its postfire production often increases when competition from other plants is removed (Daubenmire 1970, as cited in Bradley 1986a). The stems of basin wildrye are coarse, and its reproductive buds are located at or just below the soil surface so most plants survive fire (McMurray 1987c).

Prairie junegrass has a small bunch and is coarse textured, so it is considered to be one of the most fire-resistant bunchgrasses. Prairie junegrass also readily recovers by establishment of seedlings (Volland and Dell 1981, as cited in Tirmenstein 1987d). Squirreltail has little fuel at its base except in situations where it has not been grazed in a long time, and its buds are about 2 inches below the soil surface, so it is rarely harmed by fire (Ahlenslager 1986a). Western wheatgrass almost always is enhanced by prescribed fire, because it has little concentration of surface fuel and its buds are dispersed along rhizomes in soil, which are very unlikely to be heated to lethal temperatures (Ahlenslager 1986a).

Muttongrass (Fendler bluegrass), found at higher elevations in the sagebrush zone, is more likely to be damaged by fire than the previous species discussed because its leaves are densely clustered, with fine to medium texture, and thus are more likely to concentrate heat at basal growth points during a fire. However, surviving individuals produce heavy seed crops after fire, so the species can reestablish in this manner (Young 1983, as cited in Winkler 1987c). Needle-and-thread grass, Thurber needlegrass, and Idaho fescue are the dominant grasses that are most easily harmed by fire in this analysis region (Tirmenstein 1987h, Tirmenstein 1987i, Bradley 1986c). All these plants have an accumulation of dense culms at their base, which tend to concentrate heat if the fire occurs during a dry period, although Thurber needlegrass has somewhat less density of basal fuel. Large-

diameter bunches of these three species have been reported to sustain more damage from fire than smaller diameter bunches. Needlegrass species have been observed to reproduce from seed after fires.

Any of these bunchgrass plants can be killed by fire. The greatest amount of damage to these plants occurs either if they are burned when actively growing or have green tissue (a time when they are more sensitive to fire temperatures) or if a fire occurs during a time when basal material is very dry, can ignite and smolder, and can concentrate heat. Prescribed fires with an objective of enhancing or maintaining grasses would not be scheduled during periods when key species are more sensitive to fire. Bunchgrass plants that survive a fire can return to preburn coverage and production within 2 years (West and Hassan 1985), but the recovery time may be shorter or much longer, depending on the amount of damage sustained by the plant, its recovery potential, site productivity, postfire weather, and postfire animal use.

In the cheatgrass zone of southeastern Oregon, southwestern Idaho, and northern Nevada, increases in this undesirable annual are possible after fire on sites that have an existing component of cheatgrass within the community. If sites with this potential are prescribed burned, perennial grasses, forbs, and shrubs are usually seeded on the site after the fire. In areas of the sagebrush region where the existing native perennial community is healthy, cheatgrass invasion after a prescribed fire is generally not a problem because native plants can compete vigorously with any cheatgrass plants that do establish. However, if these sites are overgrazed after a fire, cheatgrass may invade and eventually become dominant.

Forbs

Perennial forbs generally respond better than bunchgrasses to burning (Britton and Ralphs 1978), because their growing points are protected by soil layers to a greater extent than grasses. Fall burning does not harm most forbs because many of them are dry and disintegrated by this time (Wright, 1985). Balsamroot has been observed to respond very well to a summer wildfire after drought conditions, because it sprouts each year from well below the soil surface (Miller 1987). Forbs that are still green are very susceptible to fall fires (Wright 1985), as are such forbs as *Antennaria* spp. and *Phlox* spp. (Pechanec and Stewart 1944), which have growth points right at the surface. Perennial forbs can recover

from summer burning in 1 year (West and Hassan 1985).

Desert Shrub Analysis Region

Vegetation treatments often are not practiced on salt desert shrub, blackbrush, or Mohave and Sonoran desert shrublands (Jordan 1981), and those attempted have met with limited success. Historically, fire frequency in these vegetation types has been low. However, wildfire incidence has increased in some of these areas because of the presence of exotic annual grasses (Lotan et al. 1981, Patten and Cave 1984). Many areas of the Mohave and Sonoran Deserts are too dry during most years to produce enough fuel to carry a fire. Fires occur in the Sonoran Desert northeast of Phoenix only after 2 years of above-average precipitation, which encourages growth of annuals (Rogers and Vint 1987). Creosotebush communities rarely burn because of low herbaceous cover (Sampson and Jesperson 1963, as cited in Korthuis 1988b).

Many shrubs, trees, and cacti of the hot desert can be severely affected by burning because they are not adapted to fire. Paloverde, burroweed, bursage, broom snakeweed, ocotillo, and creosotebush are examples of desert species that can suffer high mortality rates from burning (Wright and Bailey 1982), although higher mortality rates seem associated with fires that occur under more extreme burning conditions. Creosotebush susceptibility to fire is apparently highest in June, and it has been reported to sprout after fires during other times of the year. Large numbers of triangleleaf bursage seedlings have been reported after fires in Arizona (Rogers and Steele 1980, as cited in Korthuis 1988a), and broom snakeweed can rapidly reestablish from light, wind-dispersed seed after a fire (Young 1983, as cited in Tirmenstein 1987c).

The effect of fire on Joshua trees can be quite variable. These trees are capable of producing vigorous stump and root sprouts after fire and, when moisture is adequate, may produce dense stands (Lotan et al. 1981). Observations of a southern Nevada July wildfire show that plants that seemed to sustain more physical damage during the fire did not sprout, while other plants had vigorous shoots at the base. Some plants had sprouted, but the sprouts had subsequently died (Miller 1988). Because of increases in nonnative annuals in these communities, fire frequencies are higher than they would naturally have been. The likelihood of reestablishing Joshua trees from seed is greatly reduced because younger trees are

more susceptible to fire damage than older trees. Mohave yucca sprouts prolifically from roots or nodules at the stem base after all aboveground parts of the plants are consumed by fire, with stands sometimes increasing in density (Tratz and Vogl 1978, as cited in Tirmenstein 1989d).

The following species occur in both Mohave Desert and cold desert shrub types. Shadscale, four-wing saltbush (Wright 1980), black greasewood (Young 1983, as cited in Tirmenstein 1987d), and winterfat (Dwyer and Pieper 1967) all have been reported to resprout vigorously after a fire, although August and September wildfires in southwest Idaho killed 95 to 100 percent of winterfat plants (Pellant and Reichert, as cited in Holifield 1987a). These southwest Idaho shrub communities may be somewhat atypical of winterfat communities because they are so far north in their distribution. Cool season grasses predominate, summer precipitation is rare, and grasses are usually dormant for long periods of the summer and thus are flammable, as compared to warm-season dominated communities to the south where green-up is maintained or occurs intermittently all summer in response to showers (Pellant 1989). Winterfat is reported to have good tolerance for fire when dormant (Wasser 1982, as cited in Holifield 1987a). Four-wing saltbush also has been reestablished successfully from seed after a fire in central Utah (Clary and Tiedemann 1984, as cited in Tirmenstein 1986d). Spiny hopsage, a resident of both hot and cold deserts, generally resprouts after being burned and is least susceptible to fire during summer dormancy (Rickard and McShane 1984, as cited in Holifield 1987c).

Bud sagebrush and black sagebrush, both species of the cold desert, do not sprout after fire and must recover by development of new seedlings from seed blown or carried from off the burned area (Hickerson 1986, McMurray 1987c). Black sagebrush communities rarely burn because the sparse vegetation usually precludes fire (Winward 1985, as cited in McMurray 1987c). Horsebrush and rabbitbrush both sprout after fire; their response was discussed in greater detail in the section, Sagebrush Analysis Region earlier in this appendix.

Southwestern Shrubsteppe Analysis Region

Trees and Shrubs

The most common use of fire in southwest shrubsteppe areas is to control woody species, such as snakeweed, burroweed, and creosotebush, which are fairly sensitive to burning, and velvet mesquite. While high-percentage kills of velvet mesquite are rare, even after severe wildfires (Wright and Bailey 1982), younger trees are more susceptible to fire than older trees, as are plants growing in areas with great amounts of grassy fuels (Wright 1980). Summer fires are more damaging to velvet mesquite than winter fires (Blydenstein 1957, as cited in Wright 1980). Repeated fires are necessary to maintain a grassland aspect on some prescribed fire sites, to prevent additional establishment from seeds produced by maturing sprouts of velvet mesquite and creosotebush. Honey mesquite seedlings are very tolerant of intense fires by the time they are 3 1/2 years old, so control of this species with fire is difficult (Wright et al. 1976a, as cited in Wright and Bailey 1982).

Ocotillo, Wheeler sotol, larchleaf goldenweed, and paloverde can be severely damaged by fire (Wright and Bailey 1982), as are many cactus species (Martin 1983). It has been noted that catclaw acacia is killed by fire (Thornber 1907, as cited in Wright 1980), but no additional research has been done concerning this species. Banana yucca sprouts from roots or thick, short rhizomes after fires that are not extremely severe (Tirmenstein 1989b). Soapweed yucca can sprout from underground rhizomes that are buried in soil layers. Little research data are available, but rhizomes of older soapweed yucca plants develop protective bark and are more likely to survive fires and sprout (Kingsolver 1986, as cited in Tirmenstein 1989c). As is true in other analysis regions, the amount of damage caused by fire can vary with burning conditions and timing of the fire.

Grasses

In general, perennial grasses are mildly to severely harmed by fires during dry years, but they quickly recover during wet years (Wright and Bailey 1982). Some of the species about which this has been said include Arizona cottontop, tanglehead, and hairy grama, which resembles a bunchgrass in the southern part of its range. Wolf plants of Lehmann lovegrass, an introduced bunchgrass, are severely harmed by hot June wildfires, but they were relatively unaffected by a February fire, possibly because high winter moisture content prevented burning of the crown (Pase 1971, as cited in Wright and Bailey 1982). Burning may stimulate the emergence of Lehmann lovegrass seedlings (Ruyle et al. 1988).

Burning has the greatest benefit to tobosa, big sacaton, and alkali sacaton ranges, although tobosa grass communities may require 3 years of normal precipitation to recover from a fire during a dry year (Dwyer 1972). Tobosagrass readily recovers after most fires by sprouting from extensive rhizomes and basal root crowns (Uchytil 1988b). Of the dominant perennial grasses, black grama is most seriously affected by burning because it is a stoloniferous grass, with growing points right at or near the surface. Postfire recovery is slow and hindered by postfire drought (Canfield 1939, Reynolds and Bohning 1956). If a postfire drought period is confounded by moderate grazing, black grama may never achieve preburn status in a community (Canfield 1939). In areas where annual precipitation is higher, black grama is not excessively damaged even by hot summer fires (Wright and Bailey 1982).

Chaparral Mountain Shrub Analysis Region

The ecological effects of fire in chaparral mountain shrub communities are difficult to generalize because of the diversity of this community type. Chaparral communities have evolved with fire and have developed vegetative adaptations to recurrent fire. Prescribed fire can be used to manage stands of dense chaparral, not only to reduce fuels but also to improve watershed conditions and encourage the growth of many species of palatable forage and browse species. After a fire, chaparral shrubs may sprout, reproduce from seed, or both, but without fire, sprouting shrubs will be greatly reduced in the community and replaced by those that reproduce from seed (Keeley and Zedler 1978).

Shrub live oak (turbinella oak) is the dominant species in many stands of Arizona chaparral, resprouts vigorously from root crowns after most fires (Davis and Pase 1977, as cited in Tirmenstein 1988b), and also can sprout from adventitious buds on its roots. Fuels frequently are limited in shrub live oak communities, and it is difficult to get a fire to carry through a stand (Pond and Cable, as cited in Tirmenstein 1988b). Scrub oak, western and hairy mountain mahogany, and leather oak are sprouting species that are

enhanced by burning (Keeley and Zedler 1978, Wright and Bailey 1982). Skunkbush sumac can produce sprouts from woody rhizomes or from the root crown protected by soil when aboveground vegetation is consumed by fire (Wasser 1982, as cited in Tirmenstein 1988d). In Arizona chaparral communities, root sprouting of sumac may be delayed for up to 1 year after a fire that could promote survival of at least some shoots on extremely harsh sites (Sanford 1970, as cited in Tirmenstein 1988d).

Fire heat or materials leached from charred wood enhance seed germination of many native species of chaparral communities. Point leaf manzanita and desert ceanothus maintain themselves by prolific seedling growth following prescribed fires (Keeley and Zedler 1978). Shrub cover in Arizona chaparral has been reported to reach 76 percent of preburn cover in 6 years, but it may take more than 11 years to reach preburn levels (Pase and Pond 1964, Hibbert et al. 1974, both as cited in Wright and Bailey 1982). Yield of grasses were maintained for 5 to 7 years after a burn, while forbs peaked in 2 to 3 years and rapidly declined (Pase and Pond 1964, as cited in Wright and Bailey 1982).

The dominant plant of the mountain shrub community is Gambel oak, which can resprout vigorously after fire, both from lignotubers and from rhizomes. However, wildfires can greatly decrease vigor and growth of postfire sprouts where considerable amounts of soil heating occur. In some areas where fires have burned with less severity, indicated by the presence of residual stem bases, shrubs sprout vigorously, reaching heights of 6 feet in 6 years (Zimmerman 1989).

True mountain mahogany is reported to be very fire tolerant, resprouting from root crowns even after fairly severe fires (Blauer et al. 1975, as cited in McMurray 1986d). It also reestablishes from wind- dispersed seed. In Colorado, true mountain mahogany does not sprout as prolifically as reported for other areas and can be killed if a long duration fire occurs on a site where there is a lot of litter beneath mahogany plants (Zimmerman 1989). Mature curl-leaf mountain mahogany plants are fairly sensitive to fire because sprouts only originate from buds beneath bark or from buds in branch axils. Even trees with thick bark can only survive fires that occur in sparse fuels, and shrub-like plants are usually killed (Gruell 1985, as cited in McMurray 1986a). Plants can readily establish from seed after a fire. Fire is important to maintain curl-leaf mountain mahogany, however, as plants tend to lose their ability to produce seed as they age.

Chokecherry prolifically resprouts after fire from buds on both rhizomes and root crowns, and postfire densities often increase (McMurray 1987a). Most authors report that cliffrose is severely damaged or killed by fire, although Wright et al. (1979, as cited in Holifield 1987b) report it as being a strong sprouter, and Neuenschwander (as cited in Holifield 1987b) stated that it resprouts well in western Nevada. Its sprouting ability may vary geographically because of genetic differences. Serviceberry can sprout prolifically from its root crown if the fire is not so severe as to kill all dormant buds (Miller 1987) and may sprout from more deeply buried rhizomes if the root crown is killed by the fire (Bradley 1984). Buds of mountain snowberry are located on the root crown about 1 inch below the surface, so these plants can generally sprout after low-severity fires, but they may be killed by high-severity fires that lethally heat the reproductive buds (McMurray 1986e).

A major objective for burning mountain shrub communities is to resize them, making browse more palatable for wildlife, and to increase accessibility by reducing shrub thickets. Wright and Bailey cite several authors who feel that oakbrush communities should not be burned because herbage yield and species composition are not improved unless they are artificially seeded. Sites burned in west central Colorado not only have vigorous resprouts after August prescribed fires, but also have shown excellent recovery of elk sedge (Zimmerman 1989). While some of the species of mountain shrub communities might be harmed by fires that occur under extremely dry conditions, most prescribed fires would be designed to enhance sprouting or establishment of new individuals from seed.

Pinyon-Juniper Analysis Region

Trees

Of all of the dominant pinyon and juniper species in this analysis region, alligator juniper is the only one that will reproduce vegetatively after a fire. After some fires it can resprout from the base, from shallow roots, or from buds located beneath bark along branches or the trunk. Large-diameter trees seem to lose their sprouting ability (Bassett 1987, as cited in Tirmenstein 1988a). Trees as young as 1 year old can sprout, so even though young trees are fairly susceptible to fire, managing these sites with fire is difficult.

Postfire recovery of five of the six species of pinyon and juniper depends on seed reproduction, and thus the rate of reinvasion

depends on distance to seed source, the size of the burned area, and the presence of dispersal agents. Pinyons and junipers do not produce seed until they are about 20 to 30 years old. Seeds are dispersed by birds and animals. Pinyon seeds will not germinate unless they are buried in litter or soil, so rodent caches are an extremely important postfire seed source. Seed of oneseed juniper may remain viable for a long time while buried in the soil (Johnsen 1959, as cited in Tirmenstein 1989a).

Young pinyon and juniper trees, up to about 4 feet high, are usually killed by fire. If stands are still open with a good component of sagebrush and grasses, it is possible for fire to run through it and kill the trees (Bruner and Klebenow 1978). Older trees generally become more fire resistant as bark thickens and the crown becomes more open, and they may be able to survive low-intensity fires. Large pinyon and juniper trees require fairly heavy accumulations of fine fuel beneath their canopies for the crown to be ignited (Jameson 1962). It is difficult to kill trees in fairly closed stands of pinyon-juniper because there is little live or dead fuel on the surface, and a fire will not carry unless there are extremely high winds—a situation where risk of escape of a prescribed fire is high.

A normal treatment in pinyon-juniper stands is to chain or manually cut the trees, leave the slash scattered, wait several years for grasses and shrubs to recover, and then burn the site. This removes most of the dead fuel, greatly reduces the fire hazard, and kills any residual or newly germinated pinyon and juniper trees. If a site is mechanically or manually treated only, it will probably have enhanced forage and browse production for approximately 20 years. Prescribed burning the site about 3 to 5 years after treatment, once an understory has established, will maintain the productive character of the site for about 50 years (West 1979, as cited in Tirmenstein 1986e, Wright et al. 1979, as cited in McMurray 1986b). Understory recovery in pinyon stands is very closely related to the type and number of residual plants on the site (McMurray 1986b, McMurray 1986c). If tree dominance has seriously depleted remnant shrub, forb, and grass plants, along with the soil seed reserve, the site will have to be artificially reseeded after fire (McMurray 1986b), particularly in areas where invasion by annual grasses is possible. If high rates of forage use (which reduces fuels) and fire exclusion continue to be practiced on sites invaded by pinyon juniper, tree density will continue to increase, and pinyon and juniper will continue to expand

onto shrub- and grass-dominated sites (Burkhardt and Tisdale 1976). An active management program that includes prescribed fire is necessary to reduce the amount of tree encroachment and maintain the character and productivity of the original plant community.

Shrubs

Western serviceberry, true mountain mahogany, chokecherry, winterfat, fourwing saltbush, rabbitbrush, and horsebrush are sprouting shrubs found in association with pinyon-juniper communities that have been observed to resprout vigorously after the fire (Wright et al. 1979). Bitterbrush, broom snakeweed, and curl-leaf mountain mahogany also are sprouting shrubs, but are reported to be more sensitive to burning than the previously listed species (Klebenow et al. 1976, Wright 1979). Depending on plant vigor, location of dormant buds, burning conditions, postfire weather, and site conditions, any of these species might sprout or be killed. Big sagebrush and black sagebrush are nonsprouting species that can reestablish quickly from seed (Wright and Bailey 1982).

Grasses and Forbs

Burning grasses of pinyon-juniper sites results in responses similar to those seen in sagebrush communities. Large-diameter, fine-leaved bunchgrasses are more affected than small bunchgrasses with coarse stems, and rhizomatous species tolerate fire well (Everett 1987b). Perennial forbs, except mat-forming species, such as Antennaria species, are generally only slightly damaged by fire (Wright and Bailey 1982, Everett 1987b).

Cheatgrass may increase after burning in these communities if it is present in the stand or in the area before burning, if few residual native bunchgrass plants remain on the site, or if good postfire grazing management practices are not followed. If bunchgrass communities are in good condition when the site is treated, cheatgrass may persist for only a few years. On some sites, cheatgrass may never appear (Klebenow et al. 1976).

Mountain/Plateau Grasslands Analysis Region

The effect of fire on many of the dominant shrubs and grasses in the mountain/plateau grasslands analysis region was discussed in some detail in the Sagebrush Analysis Region section, earlier in this appendix. Species covered in that section include big sagebrush, rabbitbrush, horsebrush, western wheatgrass,

bluebunch wheatgrass, rough fescue, Idaho fescue, and needle-and-thread grass. The literature does not indicate any significant differences in fire effects for these species that are characteristically related to the analysis region in this section, so the information will not be repeated here.

Shrubs

Other important shrubs of the mountain/plateau grasslands include silver sagebrush, fringed sagebrush, shrubby cinquefoil, and prickly pear cactus. Plains and mountain silver sagebrush are different than most sagebrush species in that they are moderately resistant to fire, being able to produce sprouts from roots and rhizomes. Sprouting decreases as fire severity and heat penetration into the soil increases, particularly after fall fires when the soil is dry. Silver sagebrush rapidly regains preburn cover after spring fires, although coverage is decreased significantly after many fall fires (McMurray 1987a, McMurray 1987b).

Fringed sagebrush is reported to be a weak sprouter after fire (Wright et al. 1979, as cited in Tirmenstein 1986c), although response to fire is variable. The most beneficial effects were reported after early spring fires (Anderson and Bailey 1980, as cited in Tirmenstein 1986c), and mortality has been reported after both spring and fall fires. Fringed sagebrush is a prolific seed producer, and seed may remain viable for many years and germinate when conditions are favorable. Postfire reproduction from soil-stored seed does occur.

A range of responses to fire have been reported for shrubby cinquefoil. The plant has a wide- ranging distribution and ecotypic variability that affects its ability to sprout. Whether a particular plant sprouts after a fire apparently is related to site characteristics, season of burn, fire intensity, and burn severity. Cinquefoil produces sprouts from buds on its root crown, rhizomes, and prostrate stems that survived the fire. Survival is most often reported after spring fires. Shrubby cinquefoil can also reestablish through an abundance of wind-dispersed seed (Tirmenstein 1987e).

The effect of fire on prickly pear varies with plant height, stem moisture content, and the amount of associated fuel, because the plant itself will not burn (Humphrey 1974, as cited in Holifield 1987e). It can resprout from any surviving root crowns and by the adventitious rooting of remaining pads (Holifield 1987e). Postfire death of prickly pear often is caused

by postfire damage by insects, rodents, rabbits, and livestock, or by dehydration (Holifield 1987e).

Grasses

Important native grasses of the mountain/plateau grasslands that have not been previously discussed include rough fescue, oatgrasses, and mountain brome. Rough fescue is a large-diameter, coarse-stemmed bunchgrass that seems well adapted to periodic burning. It is susceptible to damage from fires during hot, dry weather, although it has been benefited by spring and fall prescribed fires. In areas where it has not been grazed or burned for many years, accumulations of litter may ignite and smolder for a long time after a flaming front has passed, causing significant basal bud mortality. Fescue also is particularly sensitive to burning during the active growing season (Sinton 1980, as cited in McMurray 1987e). Antos et al. (1983, as cited in McMurray 1987e) suggest that the most beneficial fire frequencies for rough fescue are approximately every 5 to 10 years. Little information is available about the response of oatgrasses to fire, although other oatgrass species in the Pacific Northwest are reported to be moderately resistant to fire. One-spike oatgrass, a densely tufted to matted perennial bunchgrass was reported to increase in basal cover after two spring prescribed fires in southwest Montana (Nimir and Payne 1978). Mountain brome, a short-lived perennial bunchgrass with shallow roots, regained 76 percent of its preburn cover within 12 weeks, as compared to a control, after one of those same spring fires studied by Nimir and Payne.

The native grass species of the Palouse grasslands of eastern Washington and Oregon and northern Idaho include bluebunch wheatgrass, Idaho fescue, and Sandberg bluegrass. They have been replaced in many locations by introduced exotics, including Kentucky bluegrass, cheatgrass, medusahead, and other bromes. Severe summer fires can kill bluebunch wheatgrass and Idaho fescue in this area, although cover of these plants was not affected by cool fires (Daubenmire 1970). Cheatgrass will continue to expand at the expense of native perennials because it is so widely established and highly flammable. It will burn when native perennials are still actively growing and much more sensitive to fire heating. Medusahead is a highly flammable exotic annual that is capable of replacing cheatgrass on many areas, particularly where soils have high clay content. It can be somewhat controlled with fire if it is burned after it is cured but before seeds are

dispersed from the stalk. Many of the seeds are destroyed, and fewer seedlings will germinate. Medusahead will then offer less competition to the seedlings of seeded grasses that are usually sown on these sites after burning (Ahlenslager 1987b).

Plains Grassland Analysis Region

Shrubs

Many of the shrubs that occur in the mixed and shortgrass prairie have been discussed elsewhere in this appendix. Effects on most of these species will be only briefly mentioned here. Rabbitbrush usually reproduces both by sprouting and from seeds after a fire, although seedling reproduction will be limited if vigor of other perennial plants is high. Snakeweed is usually killed by fire, as it is only a weak sprouter. However, it can rapidly reestablish by seed (Humphrey 1984, as cited in Tirmenstein 1987c). Fourwing saltbush resprouts vigorously after fire but also reproduces from seed. The effects on winterfat vary, but it is most likely to sprout after light-severity fires. Soapweed yucca resprouts vigorously from rhizomes. The effects of fire on silver sage and prickly pear were described in detail in the earlier description of fire effects in the mountain/plateau grasslands.

Other important species are honey mesquite, sand shinnery oak, cholla, and several species of sumac. Honey mesquite, with its exceptional ability to resprout, is almost impossible to kill by burning after it is about 1 foot tall, and even the seedlings are fairly fire tolerant (Wright et al. 1976a, as cited in Wright and Bailey 1982). Sand shinnery oak sprouts prolifically after fire, and density of stems has been reported to increase 15 percent after burning (McIlvain and Armstrong 1966, as cited in Wright and Bailey 1982). Young cholla plants can be killed by fire, but those taller than 1 foot were hardly damaged by burning in New Mexico, probably because the short grasses could not generate long enough flames to hurt the upper part of the plants (Dwyer and Pieper 1967, Heirman and Wright 1973, both as cited in Wright and Bailey 1982).

Most species of sumac are perennial shrubs or small trees with a dense network of roots and rhizomes that grow 3 to 12 inches below the soil surface. Sumac species usually produce many sprouts after a fire because fire heat cannot reach all of the rhizomes. Smooth sumac has been reported to increase in density even after severe fires (Wright 1972,

as cited in Tirmenstein 1988c). Skunkbush sumac sprouts after fire from both rhizomes and root crowns (Wasser 1982, as cited in Tirmenstein 1988d). Fragrant sumac sprouts vigorously after fire in the southern Great Plains (Wright 1972, as cited in Tirmenstein 1987f), although Jackson (1965, as cited in Tirmenstein 1987f) said that its cover was "permanently" thinned after an extremely hot fire on the Texas Panhandle. It is unclear whether sand sagebrush sprouts after a fire (Wright and Bailey 1982, as cited in Tirmenstein 1986b) or develops postfire cover by seedling development (Jackson 1965, as cited in Tirmenstein 1986b). Fire has been observed to reduce the abundance and vigor of silver buffaloberry (Wright and Bailey 1980).

Grasses and Forbs

Buffalograss and blue grama are the dominant grasses of the shortgrass prairie. Buffalograss is a stoloniferous perennial grass with buds that lie at the surface. Buds may be protected from a fire by a litter layer, if present, particularly if the layer is moist. Buffalograss has been reported to both increase and decrease after spring and fall fires. In one case, it responded to a spring fire by expanding its stolons and producing seed in the first growing season. Buffalograss seed is somewhat heat resistant because each seed is protected by a hardened burr. Of seeds with a seed coat, 52 percent germinated after 12 hours of exposure to temperatures that killed hulled seed after 30 minutes (Ahring and Todd 1977, as cited in Winkler 1987b).

Blue grama is a rhizomatous perennial grass that forms dense sod mats, particularly in the north end of its range. It is a warm season grass that is usually dormant during winter, early spring, and the hottest, driest part of the summer. If burned when dormant, it can usually reproduce by sprouting from rhizomes. It generally responds best to fire when burned in the spring when soils are moist and damage to rhizomes is minimized (White and Currie 1983c, as cited in Tirmenstein 1987b). Postfire productivity is significantly influenced by the amount of precipitation received (Wright and Bailey 1980), with no loss in herbage yield by the end of the first growing season in wet years.

Annual bluegrass and western wheatgrass may take 3 years or more to recover after fires in dry years (Wright and Bailey 1982). During years with above-normal spring precipitation, these grass species can tolerate fire with no herbage reduction following the first growing season (Wright 1974). Sand dropseed is

somewhat fire resistant because it has loosely clustered, coarse stems that transfer little heat to growth points below the surface when it is burning (Ahlenslager 1988). As is true for many other plants, it seems to be most susceptible to fire when conditions are dry. Galleta is a coarse stemmed, strongly rhizomatous grass that grows in bunches but can form an open sod (West et al. 1972, as cited in Holifield 1987d). It produces sprouts from rhizomes after fire and recovers quickly after winter burns when soil moisture is sufficient (Wright 1980). Red three-awn, Muhlenbergia species, and wolftail all are harmed by fire during dry years but tolerate fire better during wet years (Dwyer and Pieper 1967, Wright 1974). Seeds of red three-awn can tolerate up to 116 °C for up to 5 minutes (Sampson 1944, as cited in Winkler 1987a), so establishment from seed after fire is likely. Burning usually increases production of sand bluestem and switchgrass but decreases little bluestem production where these grasses occur (Wright and Bailey 1982).

Important mixed prairie grasses include tobosagrass (see the effects described earlier for the southwestern shrubsteppe region), green needlegrass, sideoats grama, prairie sandreed (reedgrass), and sand dropseed. Green needlegrass is similar to other needlegrasses in that it is fairly sensitive to fire, although the effect can be moderated by burning conditions and site characteristics. Green needlegrass is most negatively affected if a fire occurs when soils are dry or where plants are large in diameter and provide more fuel (Wright and Klemmedson 1965, as cited in Tirmenstein 1987j). Sideoats grama is most seriously damaged by fire during very dry years and is tolerant of fire during exceptionally wet years (Wright and Bailey 1980) or when it is dormant (Wasser 1982, as cited in Tirmenstein 1987a). Prairie sandreed is a strongly rhizomatous grass that is fire tolerant when dormant, and it revegetates a burned area with new shoots from rhizomes. It has responded more favorably to spring fires than to fall fires, which reduced it significantly (Lyon and Stickney 1976, as cited in Uchytil 1988a). Vine mesquite and Arizona cottontop do well after fire during periods of good soil moisture (Box, Powell, and Drawe 1967, Wink and Wright 1973).

The tolerance of forbs to burning depends on the timing of the fire relative to active plant growth (Wright and Bailey 1982). Those forbs that initiate growth after the burning season are least affected, because they have the entire growing season to recover from any injury that may have been caused by fire.

Coniferous/Deciduous Forest Analysis Region

Prescribed burning can be an effective management tool in forested vegetation communities in the West. Fire is used to reduce surface fuels on clearcuts as well as in the understories of fire-resistant trees; to remove understory reproduction in ponderosa pine, Douglas-fir, and western larch forests, which provides a fuel ladder to the overstory; to thin overstocked stands of trees; to prune lower branches from trees; to create seedbed; to reduce vegetation competition with naturally regenerated or planted conifers; to enhance forage values; to maintain and improve browse quality and quantity; and to rejuvenate old stands of deciduous trees.

Ponderosa pine and western larch are the most fire-resistant trees in the EIS area, closely followed by Douglas-fir, because of their thick bark and fairly deep-rooting habits. The long needles and large buds of ponderosa pine are fire resistant, while western larch buds are protected from fire heat because they are buried in corky wood. Fires were a natural occurrence in the understories of forests dominated singly or by mixtures of these species. Without fire, understories of ponderosa pine, Douglas-fir, larch, or grand fir can develop beneath the canopies of these forests. These trees not only compete with the overstory, but the stands tend to stagnate because there are too many trees, and the understory trees pose a serious fire hazard to the overstory because they can carry fire into their crowns. Understory burning at planned intervals is the best way to manage sites with these dominant tree species. If all fires are excluded from these forests types, which historically have high frequencies of understory fire, the result can be the eventual weakening of the stand, an increase in activity of bark beetles, and an increase in the proportion of dead trees. Fuels and bug-killed trees lead to stand-destroying fires. Many acres in the American West have had fire excluded for 50 to 75 years, and some of the fires in recent years are likely a result of the accumulation of fuels and insect activity.

Slash from thinning and selective logging can be burned to reduce the fire hazard, without harming the residual trees in these communities. Ponderosa pine is generally not clearcut, but clearcuts in Douglas-fir and western larch are often burned to manage the fuels, prepare seedbed and planting spots, and manage competing plants. Without fire, ponderosa pine and Douglas-fir sometimes invade grasslands, and prescribed fire can be

easily used to eliminate these trees when they are young.

Lodgepole pine, limber pine, any of the spruces, grand fir, and subalpine fir are fairly fire sensitive because of their thin bark and shallow roots. Subalpine fir also has an extremely flammable crown. A natural cycle in these forests is for bark beetle infestations to occur in mature or old trees, with resultant increased fire hazard because of the dead trees and an eventual stand-replacing fire. Prescribed fire has a role in managing logging slash from these forests, but prescribed underburning is not a standard practice, as many of these species can be killed by fires of even low intensity. Encroachment of these species into mountain meadows can be controlled with prescribed fire.

The relatively thin bark of aspen makes it fairly sensitive to fire, and the overstory is generally killed by fires with flames longer than approximately 2 feet if they are sustained for a minute or more (Brown and Simmerman 1986). However, the species sprouts prolifically from its root buds after burning, with maximum sprouting usually occurring after moderate severity fires (Brown and Simmerman 1986). The lack of understory herbaceous fuel caused by livestock grazing precludes the occurrence of fire in most aspen stands (Jones and DeByle 1985). Without fire, conifers invade many aspen stands, gradually eliminating the aspen, as aspen sucker replacement often is insufficient to replace overstory aspen mortality (Schier 1975). Aspen communities on sites not suited for conifer establishment may eventually be replaced by grasses and shrubs (Schier 1985). Suckering is prevented by the presence of mature trees as the trees and roots gradually deteriorate. Because of this phenomenon a loss of aspen stands has been observed in several Western States. A fire that occurs in an aspen stand that is still producing a few suckers or in a mixed aspen-conifer stand is likely to result in the rejuvenation of the aspen stand. The amount of postfire suckering is enhanced by warmer soil temperatures, which usually occur as a result of the blackened soil surface and reduced thickness of the litter and organic layer (Jones and DeByle 1985). As is true for rangeland sites, an aspen site must be rested from grazing until the community recovers to some degree (Brown and Simmerman 1986). Wildlife use can be regulated to some extent if a large enough burned area is selected or if several areas in the same general vicinity are burned, thus dispersing use over a greater acreage.

The effect of prescribed underburning in communities that contain ponderosa pine, Douglas-fir, or western larch depends on the associated tree species, the understory species present on the site, season of the fire, and fuel, duff, and soil moisture conditions at the time of the fire. Whether natural fuels or thinning slash is being burned, prescriptions can be selected that will minimize the impacts on these three conifers, because their morphology makes them fire resistant. Flame lengths can be minimized, and the amount of consumption of large-diameter fuels that could harm cambium or roots can be limited. Other, less fire-tolerant conifers, such as grand fir, could be harmed by a fire that would not hurt the ponderosa, Douglas-fir, or larch. However, if fire suppression has been effective in these forests for many years, the presence of any less fire-resistant conifers is the result of the exclusion of fire. Care will have to be taken in underburning these stands to prevent harm to the fire-resistant conifers caused by the unnatural presence of fire-sensitive conifers that may be intermixed with them.

The understories of these communities are all adapted to fire. Some later successional species that may have established because of fire exclusion might not be favored, but the natural shrub, forb, and grass associates of these species would recover by sprouting or from seed stored in the forest soil organic layer (duff) after fire. The exact response varies by fire prescription, season, moisture condition, and plant species—a topic that would be covered in a site-specific environmental assessment.

Slash burning potentially could do more harm to a site than prescribed underburning because of the presence of large amounts of slash on the soil surface. An objective for slashburning may be to kill some of the understory species, so less competition is present for trees that might be planted. Specific ranges of moisture content of large-diameter fuels, duff, and soil can be selected for the prescribed fire prescription that will have the desired effect on understory vegetation, with consideration given to the effects of burning on the soil. One obvious effect of this treatment, which is perhaps more closely associated with the removal of the forest overstory than with the burning itself, is that plants that require sunlight will do better after the treatment than those that require shade. This change in dominant species, or species present, would persist until the forest overstory again develops to the point where it provides a good cover of shade.

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Appendix G
Species Scientific Names

This appendix contains a list of the common and scientific names for wildlife and vegetation species mentioned in the text. A dagger (†) indicates the plant species that have been identified as target species in particular States. See Appendix I for this list.

Table G-1. Species Scientific Names

Common Name	Scientific Name
Mammals	
Antelope, Pronghorn	*Antilocapra americana*
Antelope, Sonoran Pronghorn	*Antilocapra americana sonoriensis*
Badger	*Taxidea taxus*
Bat, Sanborn's Long-nosed	*Leptonycteris sanborni*
Bear, Black	*Ursus americanus*
Bear, Grizzly	*Ursus arctos horribilis*
Beaver	*Castor canadensis*
Bison, American	*Bison bison*
Bobcat	*Lynx rufus*
Caribou, Woodland	*Rangifer tarandus*
Cat, Ringtail	*Bassariscus astutus*
Chipmunk, Least	*Eutamias minimus*
Coati	*Nasua nasua*
Cottontail, Desert	*Sylvilagus audubonii*
Cottontail, Pygmy	*Sylvilagus idahoensis*
Coyote	*Canis latrans*
Deer, Mule	*Odocoileus hemionus*
Deer, White-tailed	*Odocoileus virginianus*
Deer, Coves' White-tailed	*Odocoileus virginianus couesi*
Elk	*Cervus elaphus*
Ferret, Black-footed	*Mustela nigripes*
Flying Squirrel, Northern	*Glaucomys sabrinus*
Fox, Gray	*Urocyon cinereoargenteus*
Fox, Kit	*Vulpes macrotes*
Goat, Mountain	*Oreamnos americanus*
Gopher, Pocket	*Thomomys spp.*
Ground Squirrel, Columbia	*Spermophilus columbianus*
Ground Squirrel, Golden Mantled	*Spermophilus lateralis*
Jackrabbit, Black-tailed	*Lepus californicus*
Jackrabbit, White-tailed	*Lepus towsendii*
Jaguarundi	*Felis yagouaroundi tolteca*
Lion, Mountain	*Felis concolor*
Moose	*Alces alces*
Mouse, Brush	*Peromyscus boylii*
Mouse, Deer	*Peromyscus maniculatus*
Mouse, Little Pocket	*Perognathus longimembris*
Mouse, Pinyon	*Peromyscus truei*
Mouse, Southern Grasshopper	*Onychomys torridus*
Mouse, White-footed	*Peromyscus truei*
Ocelot	*Felis pardalis*
Oppossum	*Didelphis marsupialis*
Peccary, Collared	*Pecari angulatus*
Prairie Dog, Black-tailed	*Cynomys ludovicianus*
Prairie Dog, Utah	*Cynomys parvidens*
Prairie Dog, White-tailed	*Cynomys leucurus*
Raccoon	*Procyon lotor*
Rat, Bannertail Kangaroo	*Dipodomys spectabilis*
Rat, Desert Kangaroo	*Dipodomys deserti*
Rat, Desert Wood	*Neotoma lepida*
Rat, Great Basin Kangaroo	*Dipodomys microps*
Rat, Merriam's Kangaroo	*Dipodomys merriami*
Rat, Ord's Kangaroo	*Dipodomys ordii*
Rat, White-throated Wood	*Neotoma albigula*
Rat, Wood	*Neotoma sp.*
Sheep, Bighorn	*Ovis canadensis*
Sheep, Desert Bighorn	*Ovis canadensis nelsoni*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Skunk, Spotted	*Spilogale putorius*
Skunk, Striped	*Mephitus mephitis*
Squirrel, Abert's	*Sciurus aberti*
Squirrel, Harris' Antelope	*Ammospermophilus harrisii*
Squirrel, Kaibab	*Sciurus aberti kaibababensis*
Squirrel, Red	*Tamiasciurus hudsonicus*
Squirrel, Rock	*Spermophilus variegatus*
Squirrel, White-tailed Antelope	*Ammospermophilus leucurus*
Vole, Sagebrush	*Lagurus curtatus*
Wolf, Gray	*Canis lupus*

Birds

Bluebird, Western	*Sialia mexicana*
Chickadee, Mountain	*Parus gambeli*
Crane, Whooping	*Grus americana*
Cuckoo, Yellow-billed	*Coccyzus americanus*
Dove, Common Ground	*Columbina passerina*
Dove, White-winged	*Zenaida asiatica*
Eagle, Bald	*Haliaeetus leucocephalus*
Eagle, Golden	*Aquila chrysaetos*
Falcon, American Peregrine	*Falco peregrinus anatum*
Falcon, Northern Aplomado	*Falco femoralis septentrionalis*
Falcon, Prairie	*Falco mexicanus*
Finch, Cassin's	*Carpodacus cassisii*
Flicker, Northern	*Colaptes auratus*
Flycatcher, Ash-throated	*Myiarchus cinerascens*
Flycatcher, Gray	*Empidonax wrightli*
Flycatcher, Vermilion	*Pyrocephalus rubinus*
Flycatcher, Western	*Empidonax difficilis*
Goshawk, Western	*Accipiter gentilis*
Grouse, Blue	*Dendragapus obscurus*
Grouse, Ruffed	*Bonasa umbellus*
Grouse, Sage	*Centrocercus urophasianus*
Grouse, Sharp-tailed	*Pedioecetes phasianellus*
Hawk, Ferruginous	*Buteo regalis*
Hawk, Harris'	*Parabuteo unicinctus*
Hawk, Marsh	*Circus cyaneus*
Hawk, Red-tailed	*Buteo jamaicensis*
Hawk, Swainson's	*Buteo swainsonii*
Hawk, Zone-tailed	*Buteo albonotatus*
Hummingbird, White-eared	*Hylocharis leucotis*
Jay, Gray-breasted	*Aphelocoma ultramarina*
Jay, Pinyon	*Gymnorhinus cyanocephalus*
Jay, Scrub	*Aphelocoma coerulescens*
Jay, Stellar's	*Cyanocitta stelleri*
Junco, Dark-eyed	*Junco hyemalis*
Kestrel, American	*Falco sparverius*
Killdeer	*Charadrius vociferus*
Lark, Horned	*Eremophila alpestris*
Lark, Western Meadow	*Sturnella neglecta*
Longspur, McCown's	*Rhynochophanes mccownii*
Magpie, Black-billed	*Pica pica*
Nighthawk, Lesser	*Chordeiles minor*
Nutcracker, Clark's	*Nucifraga columbiana*
Nuthatch, Pygmy	*Sitta pygmaea*
Nuthatch, Red-breasted	*Sitta canadensis*
Owl, Burrowing	*Athene cunicularia*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Owl, Elf	*Micrathene whitney*
Owl, Saw-whet	*Aegolius acadicus*
Partridge, Chukar	*Alectoris chukar*
Phainopepla	*Phainopepla nitens*
Pigeon, Band-tailed	*Columba fasciata*
Plover, Piping	*Charadrius melodus*
Prairie-Chicken	*Tympanuchus spp.*
Pyrrhuloxia	*Cardinalis sinuatus*
Quail, Gambel's	*Callipepla gambelii*
Quail, Montezuma	*Cyrontyx montezumae*
Quail, Mountain	*Oreortyx pictus*
Quail, Scaled	*Callipela squamata*
Rail, Yuma Clapper	*Rallus longirostris yumanensis*
Raven, Chihuahuan	*Corvus cryptoleucus*
Raven, Common	*Corvus corax*
Sapsucker, Williamson's	*Sphyrapicus thyroideus*
Screech-owl, Whiskered	*Otus asio*
Shrike, Loggerhead	*Lanius ludovicianus*
Sparrow, Black-chinned	*Spizella atrogularis*
Sparrow, Sage	*Amphispiza belli*
Sparrow, Vesper	*Pooecetes gramineus*
Tanager, Western	*Piranga ludoviciana*
Tern, Least	*Sterna albifrons*
Thrasher, Bendire's	*Toxostoma bendirei*
Thrasher, Sage	*Oreoscoptes montanus*
Thrush, Swainson's	*Catharus ustulatus*
Titmouse, Bridled	*Parus wollweberi*
Titmouse, Plain	*Parus inornatus*
Towhee, Abert's	*Pipilo aberti*
Towhee, Rufous-sided	*Pipilo erythrophthalmus*
Turkey, Wild	*Meleagris gallopavo*
Verdin	*Auriparus flaviceps*
Warbler, Black-throated Gray	*Dendroica nigrescens*
Warbler, Lucy's	*Vermivora luciae*
Woodpeewee, Western	*Contopus sordidulus*
Woodpecker, Acorn	*Melanerpes formicivorus*
Woodpecker, Strickland's	*Picoides stricklandi*
Wren, Bewick's	*Thryomanes bewickii*
Wren, Cactus	*Campylorhynchus brunneicapillus*
Wren, Canyon	*Catherpes mexicanus*
Wren, Rock	*Salpinctes obsoletus*
Wrentit	*Chamaea fasciata*
Reptiles and Amphibians	
Bullfrog	*Rana catesbeiana*
Bullsnake	*Pituophis sp.*
Frog, Leopard	*Rana pipiens*
Frog, Rio Grande Leopard	*Rana pipiens berlandieri*
Gila Monster	*Heloderma suspectum*
Lizard, Collared	*Crotaphytus collaris*
Lizard, Horned	*Phrynosoma spp.*
Lizard, Lesser Earless	*Holbrookia maculata*
Lizard, Round-tailed Horned	*Phrynosoma modestum*
Lizard, Sagebrush	*Sceloporus graciosus*
Lizard, Southern Prairie	*Sceloporus undulatus consobrinus*
Lizard, Side-blotched	*Uta stansburiana*
Lizard, Tree	*Urosaurus ornatus*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Massasauga	*Sistrurus catenatus*
Rattlesnake, Great Basin	*Crotalis viridis*
Rattlesnake, Western	*Crotalus viridis*
Rattlesnake, Western Diamondback	*Crotalus atrox*
Salamander, Blotched Tiger	*Ambystoma tigrinum melanostictum*
Salamander, Tiger	*Ambystoma tigrinum*
Sidewinder	*Crotalus cerastes*
Skink, Great Plains	*Eumeces obsoletus*
Skink, Western	*Eumeces skiltonianus*
Snake, California King	*Lampropeltis getulus californiae*
Snake, Common Garter	*Thamnophis spp.*
Snake, Glossy	*Arizona elegans*
Snake, Night	*Hypsiglena torquata texana*
Snake, Mexican Garter	*Thamnophis eques*
Snake, Pine Gopher	*Pituophis melanoleucus*
Snake, Plains Garter	*Thamnophis radix*
Snake, Striped Whip	*Masticophis taeniatus*
Snake, Wandering Garter	*Thamnophis elegans vagrans*
Snake, Western Hognosed	*Heterodon nasicus*
Snake, Western Hooknosed	*Ficimia cana*
Snake, Western Terrestrial Garter	*Thamnophis elegans*
Spadefoot, Plains	*Scaphiopus bombifrons*
Spadefoot, Western	*Scaphiopus hammondi*
Toad, Great Plains	*Bufo cognatus*
Toad, Green	*Bufo debilis*
Toad, Wyoming	*Bufo hemiophrys baxteri*
Tortoise, Desert	*Gopherus agassizii*
Turtle, Western Box	*Terrapene ornata*
Whiptail, Desert Grassland	*Cnemidophorus uniparens*
Whiptail, Plateau	*Cnemidophorus velox*
Whiptail, Spotted	*Cnemidophorus sacki*
Whiptail, Western	*Cnemidophorus tigris*
Fish	
Bass, Largemouth	*Micropterus salmoides*
Bluegill	*Lepomis macrochirus*
Carp, Asian	*Cyprinus carpio*
Catfish, Channel	*Ictalurus punctatus*
Catfish, Yaqui	*Ictalurus pricei*
Chub, Bonytail	*Gila elegans*
Chub, Borax Lake	*Gila boraxobius*
Chub, Chihuahua	*Gila nigrescens*
Chub, Humpback	*Gila cypha*
Chub, Pahranagat Roundtail	*Gila robusta jordani*
Chub, Roundtail	*Gila robusta*
Chub, Sonora	*Gila ditaenia*
Chub, Yaqui	*Gila purpurea*
Crappie	*Pomoxis spp.*
Cul-ui	*Chasmistes cujus*
Dace, Ash Meadows Speckled	*Rhinichthys osculus nevadensis*
Dace, Desert	*Eremichthys acros*
Dace, Longfin	*Agosia chrysogaster*
Dace, Speckled	*Rhinichthys osculus*
Gambusia, Pecos	*Gambusia nobilis*
Killifish, Pahrump	*Empetrichthys latos*
Minnow	*Cyprinidae spp.*
Minnow, Loach	*Tiaroga cobitis*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Perch, Yellow	*Perca flavescens*
Pupfish, Ash Meadows Amaragosa	*Cyprinodon nevadensis mionectes*
Pupfish, Desert	*Cyprinodon macularius*
Pupfish, Devil's Hole	*Cyprinodon diabolis*
Pupfish, Warm Springs	*Cyprinodon nevadensis pectoralis*
Salmon, Coho	*Oncorhynchus kisutch*
Salmon, Pacific	*Oncorhynchus spp.*
Shad, Gizzard	*Dorosoma cepedianum*
Shiner, Beautiful	*Notropis formosus*
Spikedace	*Meda fulgida*
Spinedace, Big Spring	*Lepidomeda mollispinis pratensis*
Spinedace, White River	*Lepidomeda albivallis*
Springfish, Hiko White River	*Crenichthys baileyi grandis*
Springfish, Railroad Valley	*Crenichthys baileyi nevadea*
Springfish, White River	*Crenichthys baileyi*
Squawfish	*Ptychocheilus spp.*
Squawfish, Colorado River	*Ptychocheilus lucius*
Stoneroller, Central	*Campostoma anomalum*
Sucker	Catostomidae
Sucker, Lost River	*Deltistes luxatus*
Sucker, Warner	*Catostomus warnerensis*
Sunfish, Green	*Lepomis cyanellus*
Topminnow, Gila (Yaqui)	*Poeciliopsis occidentalis*
Trout, Apache	*Salmo apache*
Trout, Brook	*Salvelinus fontinalis*
Trout, Brown	*Salmo trutta*
Trout, Cutthroat	*Salmo clarki*
Trout, Gila	*Salmo gilae*
Trout, Lahontan Cutthroat	*Salmo clarki henshawi*
Trout, Rainbow	*Salmo gairdneri*
Trout, Steelhead	*Salmo gairdneri*
Walleye	*Stizostedion vitreum vitreum*
Woundfin	*Plagopterus argentissimus*
Invertebrates	
Ant, Harvester	*Pogonomyrmex barbatus*
Ant, Rough Harvester	*Pogonomyrmex rugosus*
Bee, Honey	*Apis mellifera*
Beetle, Desert Skunk	*Eleodes armata*
Beetle, Three-lined Potato	*Lema trilineata*
Bumblebee, Golden Northern	*Bumbus ferridus*
Butterfly, Acmon Blue	*Plebejus acmon*
Butterfly, Alfalfa Looper	*Autographa californica*
Butterfly, Becker's White	*Pontia beckerii*
Butterfly, Blue	Polyommatinae
Butterfly, California Sister	*Adelpha bredowii*
Butterfly, Chalcedona Checkerspot	*Euphdryas chalcedona*
Butterfly, Common Checkered Skipper	*Pyrgus communis*
Butterfly, Copper	Lycaena
Butterfly, Dark Zebra Swallowtail	*Eurytides philolaus*
Butterfly, Lorquin's Admiral	*Limentis lorquini*
Butterfly, Monarch	*Danaus plexippus*
Butterfly, Mylitta Cresent-spot	*Phyciodes mylitta*
Butterfly, Northwest Ringlet	*Coenonympha ampelos*
Butterfly, Orange-bordered Blue	*Lycaeides melissa*
Butterfly, Oregon Silverspot	*Speyeria zerene hippolyta*
Butterfly, Prairie Ringlet	*Coenonympha inornata*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Butterfly, Sleepy Orange	*Eurema nicippe*
Butterfly, Small Checkered Skipper	*Pyrgus scriptura*
Butterfly, West Coast Lady	*Vanessa annabella*
Butterfly, Western Sister	*Adelpha bredowii*
Butterfly, Western-tailed Blue	*Everes amyntula*
Cricket, Field	*Gryllus pennsylvanicus*
Grasshopper, Panther-spotted	*Poecilotettix pantherina*
Isopod, Soccoro	*Thermosphaeroma thermophilus*
Katydid, Broad-winged	*Microcentrum rhombifolium*
Moth, Barnes' Tiger	*Ozadamia barnesii*
Moth, Greasewood	*Agapema galbina*
Moth, Hawk	Sphingidae
Moth, Leafy Spurge Hawk	*Hyles euphorbiae*
Moth, Ornate Tiger	*Apnathesis ornata*
Moth, Tiger	Arctiidae
Naucorid, Ash Meadows	*Ambrysus amargosus*
Scorpion, Centruroides	*Centruroides* spp.
Spider, Golden Huntsman	*Olios fasciculatus*
Spider, Metaphid Jumping	*Metaphidippus* spp.
Tarantula, American	*Dugesiella hentzi*
Tarantula, Desert	*Aphonopelma chalcodes*
Wasp, White Grub	*Triscolia ardens*

Shrubs, Trees, and Succulents

Acacia	*Acacia* spp.
Agave, Arizona	*Agave arizonica*
All Scale	*Atriplex polycarpa*
Allthorn	*Koeberlinia spinosa*
Ash	*Fraxinus* spp.
Aspen, Quaking	*Populus tremuloides*
Birch, River	*Betula nigra*
Birch, Water	*Betula occidentalis*
Bitterbrush	*Purshia tridentata*
Blackbrush	*Coleogyne ramosissima*
Blackgum	*Nyssa sylvatica*
Blazingstar, Ash Meadows	*Mentzelia leucophylla*
Boxelder	*Acer negundo*
Bricklebush	*Brickellia* spp.
Broom, Desert	*Baccharis sarothroides*
Buckbrush	*Ceanothus cuneatus*
Buckwheat	*Eriogonum* spp.
Buffaloberry, Silver	*Shepherdia argentea*
† Burroweed	*Haplopappus tenuisectus*
Bursage	*Ambrosia dumosa* and *A. deltoidea*
Cacti, Arizona Hedgehog	*Echinocereus triglochidiatus* var. *arizonicus*
Cacti, Barrel	*Ferocactus* spp.
Cacti, Brady Pincushion	*Pediocactus bradyi*
Cacti, Bunched Cory	*Coryphantha vamillosa*
Cacti, Hedgehog	*Echinocereus* spp.
Cacti, Knowlton	*Pediocactus knowltoni*
Cacti, Kuenzler Hedgehog	*Echinocereus fendleri* var. *kuenzleri*
Cacti, Lee Pincushion	*Coryphantha sneedii* var. *leei*
Cacti, Mesa Verde	*Sclerocactus mesae-verdae*
Cacti, Nellie Cory	*Coryphanta minima*
Cacti, Peebles Navajo	*Pediocactus peeblesianus* var. *peeblesianus*
Cacti, Prickly Pear	*Opuntia* spp.
Cacti, San Rafael	*Pediocactus despainii*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Cacti, Sneed Pincushion	*Coryphanta sneedii* var. *sneedii*
Cacti, Spineless Hedgehog	*Echinocereus triglochidiatus* var. *inermis*
Candelilla	*Euphorbia antisyphilitica*
† Ceanothus	*Ceanothus* spp.
Chokecherry	*Prunus virginiana*
† Cholla	*Opuntia* spp.
Cinquefoil (shrubby)	*Potentilla* spp.
Cliffrose	*Cowania mexicana*
Cliffrose, Arizona	*Cowania subintegra*
Cottonwood	*Populus fremontii; Populus trichocarpa; Populus angustifolia*
† Creosotebush	*Larrea tridentata*
Currant	*Ribes* spp.
Cycladenia, Jones	*Cycladenia humilis* var. *jonesii*
Cypress, Bald	*Taxodium distichum*
Dogwood	*Cornus stolonifera*
† Elderberry	*Sambucus canadensis*
Elm	*Ulmus* spp.
Fir, Douglas	*Pseudotsuga menziesii*
Fir, Grand	*Abies grandis*
Fir, Subalpine	*Abies lasiocarpa*
Fir, White	*Abies concolor*
Grass, Bear	*Nolina* spp.
† Greasewood	*Sarcobatus vermiculatus*
Hackberry	*Celtis reticulata*
Hawthorn, Black	*Crataegus douglasii*
Hemlock, Western	*Tsuga heterophylla*
Hollyleaf Buckthorn	*Rhamnus crocea*
Hopsage, Spiny	*Grayia spinosa*
Horsebrush	*Tetradymia* spp.
Indigobush	*Dalea* spp.
Ironweed	*Olneya tesota*
Ironwood	*Vernonia* spp.
Javelina-bush	*Condalia ericoides*
Jojoba	*Simmondsia chinensis*
Joshua-tree	*Yucca brevifolia*
Juniper, Alligator	*Juniperus depeana*
† Juniper, One-seed	*Juniperus monosperma*
Juniper, Rocky Mountain	*Juniperus scopulorum*
Juniper, Utah	*Juniperus osteosperma*
Juniper, Western	*Juniperus occidentalis*
Kinnikinnick	*Arctostaphylos uva-ursi*
Larch, Western	*Larix occidentalis*
Lavendar, Desert	*Hyptis emoryi*
Lechuguilla	*Agave lechuguilla*
Locust, Black	*Robinia pseudoacacia*
Locust, Honey	*Gleditsia triacanthos*
Locust, New Mexico	*Robinia neomexicana*
Manzanita, Pointleaf	*Arctostaphylos pungens*
Manzanita, Pringle	*Arctostaphylos pringlei*
Maple, Big-tooth	*Acer grandidentatum*
† Mesquite	*Prosopis juliflora* and *P. velutina*
Mesquite, Honey	*Prosopis glandulosa*
Mesquite, Velvet	*Prosopis velutina*
Mimosa	*Mimosa* spp.
Mormon Tea	*Ephedra* spp.

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Mountainmahogany	*Cercocarpus montanus, Cercocarpus ledifolius, and Cercocarpus betuloides*
† Mulberry	*Morus* spp.
† Nine-bark	*Physocarpus malvaceus*
Oak, Emory	*Quercus emoryi*
† Oak, Gambel	*Quercus gambelii*
Oak, Gray	*Quercus grisea*
† Oak, Shrub Live	*Quercus turbinella*
Ocotillo	*Fouquieria* spp.
Palo Verde, Blue	*Cerdidium floridum*
Pickleweed	*Allenrolfea occidentalis*
Pine, Bristle Cone	*Pinus aristata*
Pine, Limber	*Pinus flexilis*
Pine, Lodgepole	*Pinus contorta*
Pine, Ponderosa	*Pinus ponderosa*
Pine, Western White	*Pinus monticola*
Pine, White Bark	*Pinus albicaulis*
Pinyon, Doubleleaf	*Pinus edulis*
Pinyon, Mexican	*Pinus cembroides*
Pinyon, Singleleaf	*Pinus monophylla*
Plume, Apache	*Fallugia paradoxa*
Rabbitbrush, Big	*Chrysothammus nauseosus*
Rabbitbrush, Little	*Chrysothamnus viscidiflorus*
Redcedar, Western	*Thuja plicata*
Rose, Wild	*Rosa* spp.
Sagebrush, Basin Big	*Artemisia tridentata* spp. *tridentata*
† Sagebrush, Big	*Artemisia tridentata*
Sagebrush, Black	*Artemisia nova*
Sagebrush, Fringed	*Artemisia frigida*
Sagebrush, Low	*Artemisia arbuscula*
Sagebrush, Mountain Big	*Artemisia tridentata* spp. *vaseyana*
† Sagebrush, Sand	*Artemisia filifolia*
Sagebrush, Silver	*Artemisia cana*
Sagebrush, Wyoming Big	*Artemisia tridentata* spp. *Wyomingensis*
Saguaro	*Cereus giganteus*
† Saltbush, Fourwing	*Atriplex canescens*
Saltbush, Gardner	*Atriplex gardneri*
Saltbush, Mat	*Atriplex corrugata*
† Saltcedar (tamarisk)	*Tamarix pentandra*
Seepweed	*Suaeda* spp.
Seepwillow	*Baccharis salicifolia*
Serviceberry, Western	*Amelanchier alnifolia*
Shadscale	*Atriplex confertifolia*
Silktassel, Yellowleaf	*Garrya Flavescens*
† Snakeweed ·	*Gutierrezia sarothrae*
Snowberry, Common	*Symphoricarpos oreophilus*
Sophora	*Sophora* spp.
Sotol	*Dasylirion* spp.
Spruce, Blue	*Picea pungens*
Spruce, Englemann	*Picea engelmannii*
Sumac	*Rhus* spp.
Sweetgum	*Liquidambar* spp.
Sycamore	*Platanus* spp.
† Tarbush	*Flourensia cernua*
Thornbush	*Lycium andersonii*
Walnut	*Juglans* spp.
† Weed, Klamath	*Hypericum perforatum*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Whortleberry, Grouse	*Vaccinium scoparium*
Willow	*Salix* spp.
Willow, Desert	*Chilopsis linearis*
Winterfat	*Eurotia lanata*
Yucca	*Yucca* spp.
Forbs	
Alkali Weed	*Nitrophila* spp.
Arrowweed	*Pluchea sericea*
Aster	*Aster* spp.
Balsamroot	*Balsamorhiza sagittata*
† Bindweed, Field	*Convolvulus arvensis*
† Bindweed, Hedge	*Convolvulus sepium*
Cinquefoil	*Potentilla* spp.
† Cocklebur	*Xanthium* sp.
† Cocklebur, Mexican	*Xanthium pennsylvanicum*
Coneflower	*Ratibida columnaris*
Dandelion	*Malacothrix* spp.
Dogweed	*Dyssodia* spp.
Evening Primrose	*Oenothera* spp.
Filaree	*Erodium cicutarium*
Fleabane	*Erigeron* spp.
Fleabane, Zuni	*Erigeron rhizomatus*
† Foxtail, Giant	*Setaria faberi*
Geranium	*Geranium* spp.
Glasswort	*Salicornia* spp.
Goldeneye	*Viguiera* spp.
Groundsel	*Senecio* spp.
Groundsel, San Francisco Peaks	*Senecio franciscanus*
Gumplant, Ash Meadows	*Grindelia fraxino-pratensis*
† Gumweed, Curly Cup	*Grindelia squarrosa*
† Halogeton	*Halogeton glomeratus*
† Hemlock, Poison	*Conium maculatum*
† Hounds Tongue	*Cynoglossum officinale*
Indian Blanket	*Gaillardia pulchella*
† Iris	*Iris* spp.
Klamathweed	*Hypericum perforatum*
† Knapweed	*Centaurea* spp.
† Kochia	*Kochia scoparia*
Larkspur	*Delphinium* spp.
Lily, Sego	*Calochortus nuttallii*
Lily, Star	*Leucocrinum montanum*
Locoweed	*Oxytropis* spp. and *Astragalus* spp.
Lupine	*Lupinus* spp.
Mallow, Globe	*Spaeralcea* spp.
Marigold, Marsh	*Caltha leptosepala*
Mariola	*Parthenium incanum*
† Milkweed, Welsh's	*Asclepius welshii*
Mules Ear	*Wyethia amplexicaulis*
† Mustard, Tumble	*Sisymbrium altissimum*
† Nettle, Horse	*Solanum caroliense* and *S. elaeagnifolium*
† Nightshade	*Solanum* spp.
Onion, Wild	*Allium* spp.
Paintbrush, Indian	*Castilleja* spp.
Parsnip, Cow	*Heracleum lanatum*
Pennyroyal, McKittrick	*Hedeoma apiculatum*
Pennyroyal, Todsen's	*Hedeoma todsenii*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Penstemon	*Penstemon* spp.
Phlox	*Phlox* spp.
Ragweed	*Ambrosia* spp.
Rubberweed	*Hymenoxys richardsonii*
Scurf Pea	*Psoralea tenuiflora*
Spurge	*Euphorbia* spp.
† Spurge, Leafy	*Euphorbia esula*
† Starthistle, Yellow	*Centaurea solstitialis*
† Sunflower	*Helianthus annuus*
† Thistle, Blue	*Echium vulgare*
† Thistle, Canada	*Circium arvense*
† Thistle, Musk	*Carduus nutans*
† Thistle, Russian	*Salsola kali*
Verbena, Desert Sand	*Abronia villosa*
† Vine, Puncture	*Tribulus terrestris*
† Woad, Dyers	*Isatis tinctoria*
Yarrow, Common	*Achillea millefolium*
Zinnia, Desert	*Zinnia acerosa*
Grasses	
Bentgrass	*Agrostis* spp.
† Bluegrass, Annual	*Poa annua*
Bluegrass, Cusick's	*Poa cusickii*
Bluegrass, Kentucky	*Poa pratensis*
Bluegrass, Sandberg	*Poa secunda*
Bluestem, Big	*Andropogon gerardii*
Bluestem, Cane	*Bothriochloa barbinoides*
Bluestem, Little	*Schizachyrium scoparium*
Bluestem, Sand	*Andropogon hallii*
Brome, Japanese	*Bromus japonicus*
Brome, Rattlesnake	*Bromus briziformis*
Brome, Red	*Bromus rubens*
Broomsedge	*Andropogon virginicus*
Buffalograss	*Buchloe dactyloides*
† Cheatgrass	*Bromus tectorum*
Chess, Soft	*Bromus mollis*
Cottontop, Arizona	*Digitaria californica*
Dropseed, Sand	*Sporobolus cryptandrus*
Fescue, Idaho	*Festuca idahoensis*
Fescue, Rough	*Festuca scabrella*
Filaree	*Erodium circutarium*
Galleta	*Hilaria jamesii*
Galleta, Big	*Hilaria rigida*
Grama, Black	*Bouteloua eriopoda*
Grama, Blue	*Bouteloua gracilis*
Grama, Hairy	*Bouteloua hirsuta*
Grama, Rothrock	*Bouteloua rothrockii*
Grama, Side-oats	*Bouteloua curtipendula*
Grama, Six-weeks	*Bouteloua barbata*
Grass, Bear	*Nolina* spp.
Grass, Pappus	*Pappophorum vaginatum*
Hairgrass	*Deschampsia* spp.
Indiangrass	*Sorghastrum nutans*
Junegrass, Prairie	*Koeleria cristata*
Lovegrass, Lehmann	*Eragrostis lehmanniana*
Lovegrass, Plains	*Eragrostis intermedia*
† Medusahead	*Taeniatherum asperum*

Table G-1. Species Scientific Names (continued)

Common Name	Scientific Name
Mesquite, Curly	*Hilaria belangeri*
Mesquite, Vine	*Panicum obtusum*
Muhly, Bush	*Muhlenbergia porteri*
Muttongrass	*Poa fendleriana*
Needle-and-Thread Grass	*Stipa comata*
Needlegrass	*Stipa spp.*
Needlegrass, Green	*Stipa viridula*
Needlegrass, Thurber	*Stipa thurberiana*
Oatgrass	*Danthonia spp.*
Reedgrass	*Calamagrostis spp.*
Ricegrass, Indian	*Oryzopsis hymenoides*
Rushes	*Juncus spp.*
Sacaton, Alkali	*Sporobolus airoides*
Saltgrass	*Distichlis spicata*
Sandreed, Prairie	*Calamovilfa longifolia*
Sedges	*Carex spp.*
Spike-rush	*Eleocharis spp.*
Squirreltail	*Sitanion hystrix*
Switchgrass	*Panicum virgatum*
Tanglehead	*Heteropogon contortus*
Threeawn	*Aristida spp.*
Tobosa	*Hilaria mutica*
Tridens, Slim	*Tridens muticus*
Wheatgrass	*Agropyron spp.*
Wheatgrass, Bluebunch	*Agropyron spicatum*
Wheatgrass, Thickspike	*Agropyron dosystachyum*
Wheatgrass, Western	*Agropyron smithii*
Wildrye, Great Basin	*Elymus cinereus*
Wolftail	*Lycurus phleoides*

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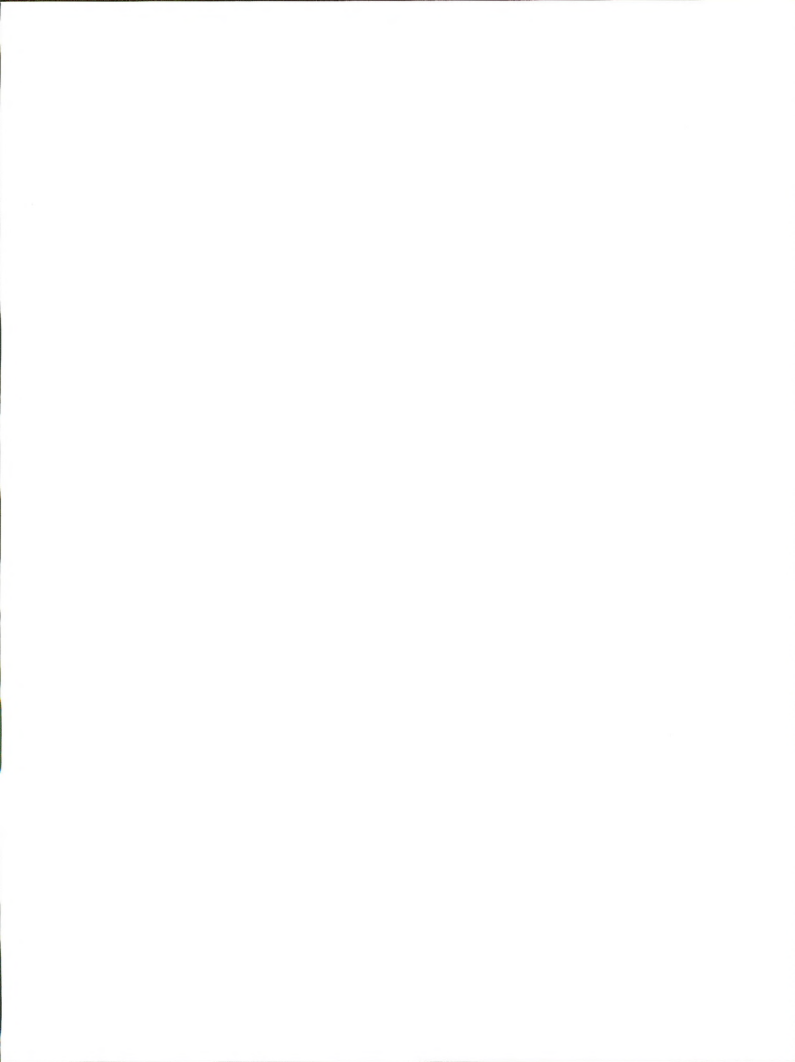
Appendix H
Special Status Species
List

This appendix is a table presenting the federally designated endangered, threatened, and candidate species found in the 13 Western States. The table has been subdivided into two parts: Part A lists only endangered and threatened species, while Part B lists candidate and proposed candidate species. Plant species in Parts A and B are alphabetized by scientific name to preserve the genera relationships for species lacking known common names. The symbols used in this table are as follows:

Part A: Endangered and Threatened Species

E = Endangered species. Any species that is in danger of extinction throughout all or a significant portion of its range.

T = Threatened species. Any species that is likely to become an endangered species within the foreseeable future throughout all or a significant portion of its range.

ECH & TCH = Endangered (E) or threatened (T) species for which a critical habitat (CH) has been designated. The term "critical habitat" is defined as the specific areas within the geographical range occupied by the species on which are found those physical or biological features essential to the conservation of the species and which may require special management considerations or protection; and specific areas outside the geographical range occupied by the species that are essential for the conservation of the species.

PE = Proposed as endangered.

PT = Proposed as threatened.

Part B: Candidate and Proposed Candidate Species

C = Candidate species. Any species for which the U.S. Fish and Wildlife Service has substantial information to support the biological appropriateness of proposing to list as endangered or threatened but has not yet issued proposed rules because of preclusion by other listing activity.

P = Proposed candidate species. Any species for which the U.S. Fish and Wildlife Service has information to propose listing it as endangered or threatened but cannot provide *conclusive* data to support the biological appropriateness of such a proposal. The taxa in this category are not being considered as proposed additions to the list unless further information becomes available.

Table H-1. Special Status Species List

Part A: Endangered and Threatened Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Mammals														
Bat, Gray	*Myotis grisescens*	—	—	—	—	—	—	—	E	—	—	—	—	—
Bat, Indiana	*Myotis solalis*	—	—	—	—	—	—	—	E	—	—	—	—	—
Bat, Mexican Long-nosed	*Leptonycteris nivalis*	—	—	—	—	—	E	—	—	—	—	—	—	—
Bat, Ozark Big-eared	*Plecotus townsendii ingens*	—	—	—	—	—	—	—	E	—	—	—	—	—
Bat, Sanborn's Long-nosed	*Leptonycteris sanborni*	E	—	—	—	—	E	—	—	—	—	—	—	—
Bear, Grizzly or Brown	*Ursus arctos (=U.a. horribilis)*	T	T	T	T	T	T	T	T	T	T	T	T	T
Caribou, Woodland	*Rangifer tarandus caribou*	—	—	E	—	—	—	—	—	—	—	—	E	—
Deer, Columbian White-tailed	*Odocoileus virginianus leucurus*	—	—	—	—	—	—	—	—	E	—	—	E	—
Ferret, Black-footed	*Mustela nigripes*	E	E	E	E	E	E	E	E	E	E	E	E	E
Jaguarundi	*Felis yagouaroundi tolteca*	E	—	—	—	—	—	—	—	—	—	—	—	—
Ocelot	*Felis pardalis*	E	—	—	—	—	—	—	—	—	—	—	—	—
Prairie Dog, Utah	*Cynomys parvidens*	—	—	—	—	—	—	—	—	—	—	T	—	—
Pronghorn, Sonoran	*Antilocapra americana sonoriensis*	E	—	—	—	—	—	—	—	—	—	—	—	—
Vole, Hualapai Mexican	*Microtus mexicanus hualpaiensis*	E	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Wolf, Gray	*Canis lupus*	E	E	E	E	E	E	E	E	E	E	E	E	E
Wolf, Red	*Canis rufus*	—	—	—	—	—	—	—	E	—	—	—	—	—
Birds														
Bobwhite, Masked (quail)	*Colinus virginianus ridwayi*	E	—	—	—	—	—	—	—	—	—	—	—	—
Crane, Whooping	*Grus americana*	E	ECH	ECH	E	—	ECH	E	ECH	—	E	E	—	E
Curlew, Eskimo	*Numenius borealis*	E	E	E	E	E	E	E	E	E	E	E	E	E
Eagle, Bald	*Haliaeetus leucocephalus*	E	E	E	E	E	E	E	E	T	E	E	T	E
Falcon, American Peregrine	*Falco peregrinus anatum*	E	E	E	E	E	E	E	E	E	E	E	ECH	E
Falcon, Arctic Peregrine	*Falco peregrinus tundrius*	T	T	T	T	T	T	T	T	T	T	T	T	—
Falcon, Northern Aplomado	*Falco femoralis septentrionalis*	E	—	—	—	—	E	—	—	—	—	—	—	—
Parrot, Thick-billed	*Rhynchopsitta pachyrhyncha*	E	—	—	—	E	—	—	—	—	—	—	—	—
Plover, Piping	*Charadrius melodus*	—	—	—	T	—	—	T	T	T	T	—	T	—
Rail, Yuma Clapper	*Rallus longirostris yumanensis*	E	—	—	—	—	—	—	—	—	—	—	—	—
Tern, Least	*Sterna antillarum*	—	E	—	E	—	E	E	E	—	E	—	—	—
Vireo, Black-capped	*Vireo atricapillus*	—	—	—	—	—	—	—	E	—	—	—	—	—
Woodpecker, Red-cockaded	*Picoides borealis*	—	—	—	—	—	—	—	E	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part A: Endangered and Threatened Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Fishes														
Catfish, Yaqui	*Ictalurus pricei*	TCH	—	—	—	—	—	—	—	—	—	—	—	—
Cavefish, Ozark	*Amblyopsis rosae*	—	—	—	—	—	—	—	T	—	—	—	—	—
Chub, Bonytail	*Gila elegans*	E	E	—	—	E	—	—	—	—	—	E	—	E
Chub, Borax Lake	*Gila boraxobius*	—	—	—	—	—	—	—	—	ECH	—	—	—	—
Chub, Chihuahua	*Gila nigrescens*	—	—	—	—	—	T	—	—	—	—	—	—	—
Chub, Humpback	*Gila cypha*	E	E	—	—	—	—	—	—	—	—	E	—	E
Chub, Hutton Tui	*Gila bicolor* ssp.	—	—	—	—	—	—	—	—	T	—	—	—	—
Chub, Pahranagat Roundtail	*Gila robusta jordani*	—	—	—	—	E	—	—	—	—	—	—	—	—
Chub, Sonora	*Gila ditaenia*	TCH	—	—	—	—	—	—	—	—	—	—	—	—
Chub, Yaqui	*Gila purpurea*	ECH	—	—	—	—	—	—	—	—	—	—	—	—
Cui-ui	*Chasmistes cujus*	—	—	—	—	E	—	—	—	—	—	—	—	—
Dace, Ash Meadows Speckled	*Rhinichthys osculus nevadensis*	—	—	—	—	ECH	—	—	—	—	—	—	—	—
Dace, Desert	*Eremichthys acros*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Dace, Foskett Speckled	*Rhinichthys osculus* ssp.	—	—	—	—	—	—	—	—	T	—	—	—	—
Dace, Kendall Warm Springs	*Rhinichthys osculus thermalis*	—	—	—	—	—	—	—	—	—	—	—	—	E
Dace, Moapa	*Moapa coriacea*	—	—	—	—	E	—	—	—	—	—	—	—	—
Darter, Leopard	*Percina pantherina*	—	—	—	—	—	—	—	TCH	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Gambusia, Pecos	*Gambusia nobilis*	—	—	—	—	—	E	—	—	—	—	—	—	—
Killifish, Pahrump	*Empetrichthys latos*	—	—	—	—	E	—	—	—	—	—	—	—	—
Minnow, Loach	*Tiaroga cobitis*	T	—	—	—	—	T	—	—	—	—	—	—	—
Pupfish, Ash Meadows Amargosa	*Cyprinodon nevadensis mionectes*	—	—	—	—	ECH	—	—	—	—	—	—	—	—
Pupfish, Desert	*Cyprinodon macularius*	ECH	—	—	—	—	—	—	—	—	—	—	—	—
Pupfish, Devil's Hole	*Cyprinodon diabolis*	—	—	—	—	E	—	—	—	—	—	—	—	—
Pupfish, Warm Springs	*Cyprinodon nevadensis pectoralis*	—	—	—	—	E	—	—	—	—	—	—	—	—
Shiner, Beautiful	*Notropis formosus*	TCH	—	—	—	—	T	—	—	—	—	—	—	—
Shiner, Pecos Bluntnose	*Notropis simus pecosensis*	—	—	—	—	—	TCH	—	—	—	—	—	—	—
Spikedace	*Meda fulgida*	T	—	—	—	—	T	—	—	—	—	—	—	—
Spinedace, Big Spring	*Lepidomeda mollispinis pratensis*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Spinedace, Little Colorado	*Lepidomeda vittata*	TCH	—	—	—	—	—	—	—	—	—	—	—	—
Spinedace, White River	*Lepidomeda albivallis*	—	—	—	—	ECH	—	—	—	—	—	—	—	—
Springfish, Hiko White River	*Crenichthys baileyi grandis*	—	—	—	—	ECH	—	—	—	—	—	—	—	—
Springfish, Railroad Valley	*Crenichthys nevadae*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Springfish, White River	*Crenichthys baileyi baileyi*	—	—	—	—	ECH	—	—	—	—	—	—	—	—
Squawfish, Colorado River	*Ptychocheilus lucius*	E^A	E	—	—	E	E	—	—	—	—	E	—	E
Sucker, June	*Chasmistes liorus*	—	—	—	—	—	—	—	—	—	—	ECH	—	—

Table H-1. Special Status Species List (continued)

Part A: Endangered and Threatened Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Sucker, Lost River	*Deltistes luxatus*	—	—	—	—	—	—	—	—	E	—	—	—	—
Sucker, Shortnose	*Chasmistes brevirostris*	—	—	—	—	—	—	—	—	—	—	—	—	—
Sucker, Warner	*Catostomus warnerensis*	—	—	—	—	—	—	—	—	—	—	—	—	—
Topminnow, Gila (incl. Yaqui)	*Poeciliopsis occidentalis*	E	—	—	—	—	E	—	—	—	—	—	—	—
Trout, Apache	*Salmo apache*	T	—	—	—	—	—	—	—	—	—	—	—	—
Trout, Gila	*Salmo gilae*	E	—	—	—	—	E	—	—	—	—	—	—	—
Trout, Greenback Cutthroat	*Salmo clarki stomias*	—	T	—	—	—	—	—	—	—	—	—	—	—
Trout, Lahontan Cutthroat	*Salmo clarki henshawi*	—	—	—	—	T	—	—	—	—	—	—	—	—
Woundfin	*Plagopterus argentissimus*	E[†]	—	—	—	E	—	—	—	—	—	E	—	—
Amphibians														
Toad, Wyoming	*Bufo hemiophrys baxteri*	—	—	—	—	—	—	—	—	—	—	—	—	E
Reptiles														
Rattlesnake, New Mexican Ridge-nosed	*Crotalus willardi obscurus*	—	—	—	—	—	TCH	—	—	—	—	—	—	—
Tortoise, Desert (Beaver Dam Slope)	*Scaptochelys (=Gopherus) agassizii*	—	—	—	—	E	—	—	—	—	—	TCH	—	—
Turtle, Leatherback Sea	*Dermochelys coriacea*	—	—	—	—	—	—	—	—	E	—	—	E	—

[†]Experimental nonessential in Gila River drainage of Arizona.

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Insects														
Butterfly, Oregon Silverspot	*Speyeria zerene hippolyta*	—	—	—	—	—	—	—	—	TCH	—	—	T	—
Naucorid, Ash Meadows	*Ambrysus amargosus*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Skipper, Pawnee Montane	*Hesperia leonardus (=pawnee) montana*	—	T	—	—	—	—	—	—	—	—	—	—	—
Crustaceans														
Isopod, Socorro	*Thermosphaeroma (=Exosphaeroma) thermophilus*	—	—	—	—	—	E	—	—	—	—	—	—	—
Plants														
Agave, Arizona	*Agave arizonica*	E	—	—	—	—	—	—	—	—	—	—	—	—
Bear-poppy, Dwarf	*Arctomecon humilis*	—	—	—	—	—	—	—	—	—	—	E	—	—
Prickly-poppy, Sacramento	*Argemone pleiacantha* ssp. *pinnatisecta*	—	—	—	—	—	PE	—	—	—	—	—	—	—
Milkweed, Welsh's	*Asclepias welshii*	—	—	—	—	—	—	—	—	—	—	TCH	—	—
Milk-vetch, Mancos	*Astragalus humillimus*	—	E	—	—	—	E	—	—	—	—	—	—	—
Milk-vetch, Heliotrope	*Astragalus limnocharis* var. *montii*	—	—	—	—	—	—	—	—	—	—	T	—	—
Milk-vetch, Rydberg	*Astragalus perianus*	—	—	—	—	—	—	—	—	—	—	T	—	—
Milk-vetch, Ash Meadows	*Astragalus phoenix*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Sedge, Navaho	*Carex specuiola*	TCH	—	—	—	—	—	—	—	—	—	—	—	—
Centaury, Spring-loving	*Centaurium namophilum* var. *namophilum*	—	—	—	—	TCH	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part A: Endangered and Threatened Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Thistle, Sacramento Mountains	Cirsium vinaceum	—	—	—	—	—	T	—	—	—	—	—	—	—
Cactus, Cochise Pincushion	Coryphantha robbinsorum (=Cochiseia r., Escobaria r.)	T	—	—	—	—	—	—	—	—	—	—	—	—
Cactus, Sneed Pincushion	Coryphantha sneedii var. sneedii	—	—	—	—	—	E	—	—	—	—	—	—	—
Cactus, Lee Pincushion	Coryphantha sneedii var. leei	—	—	—	—	—	T	—	—	—	—	—	—	—
Cliff-rose, Arizona	Cowania subintegra	E	—	—	—	—	—	—	—	—	—	—	—	—
Cycladenia, Jones	Cycladenia humilis var. jonesii	T	—	—	—	—	—	—	—	—	—	T	—	—
Cactus, Nichol's Turk's Head	Echinocactus horizonthalonius var. nicholii	E	—	—	—	—	—	—	—	—	—	—	—	—
Cactus, Arizona Hedgehog	Echinocereus triglochidiatus var. arizonicus	E	—	—	—	—	—	—	—	—	—	—	—	—
Cactus, Spineless Hedgehog	Echinocereus triglochidiatus var. inermis	—	E	—	—	—	—	—	—	—	—	E	—	—
Cactus, Purple-spined Hedgehog	Echinocereus engelmannii var. purpureus	—	—	—	—	—	—	—	—	—	—	E	—	—
Cactus, Kuenzler Hedgehog	Echinocereus fendleri var. kuenzleri	—	—	—	—	—	E	—	—	—	—	—	—	—
Cactus, Lloyd's Hedgehog	Echinocereus lloydii	—	—	—	—	—	E	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Sunray, Ash Meadows	*Enceliopsis nudicaulis* var. *corrugata*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Daisy, Maquire	*Erigeron maguirei* var. *maguirei*	—	—	—	—	—	—	—	—	—	—	—	E	—
Fleabane, Rhizome	*Erigeron rhizomatus*	—	—	—	—	—	T	—	—	—	—	—	—	—
Wild-buckwheat, Gypsum	*Eriogonum gypsophilum*	—	—	—	—	—	TCH	—	—	—	—	—	—	—
Buckwheat, Steamboat	*Eriogonum ovalifolium* var. *williamsiae*	—	—	—	—	E	—	—	—	—	—	—	—	—
Wild-buckwheat, Clay-loving	*Eriogonum pelinophilum*	—	ECH	—	—	—	—	—	—	—	—	—	—	—
Cress, Toad-flax	*Glaucocarpum suffrutescens*	—	—	—	—	—	—	—	—	—	—	E	—	—
Gumplant, Ash Meadows	*Grindelia fraxinopratensis*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Pennyroyal, McKittrick	*Hedeoma apiculatum*	—	—	—	—	—	TCH	—	—	—	—	—	—	—
Pennyroyal, Todsen's	*Hedeoma todsenii*	—	—	—	—	—	ECH	—	—	—	—	—	—	—
Ivesia, Ash Meadows	*Ivesia eremica*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Bladderpod, Dudley Bluffs	*Lesquerella congesta*	—	PT	—	—	—	—	—	—	—	—	—	—	—
Lomatium, Bradshaw's	*Lomatium bradshawii*	—	—	—	—	—	—	—	—	E	—	—	—	—
Blazingstar, Ash Meadows	*Mentzelia leucophylla*	—	—	—	—	TCH	—	—	—	—	—	—	—	—
Four-o'clock, MacFarlane's	*Mirabilis macfarlanei*	—	—	E	—	—	—	—	—	E	—	—	—	—
Cactus, Brady Pincushion	*Pediocactus bradyi*	E	—	—	—	—	—	—	—	—	—	—	—	—
Cactus, San Rafael	*Pediocactus despainii*	E	—	—	—	—	—	—	—	—	—	E	—	—

Table H-1. Special Status Species List (continued)

Part A: Endangered and Threatened Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Cactus, Knowlton	*Pediocactus knowltonii*	—	E	—	—	—	E	—	—	—	—	—	—	—
Cactus, Peebles Navajo	*Pediocactus peeblesianus* var. *peeblesianus*	E	—	—	—	—	—	—	—	—	—	—	—	—
Cactus, Siler Pincushion	*Pediocactus sileri*	E	—	—	—	—	—	—	—	—	—	E	—	—
Phacelia, Clay	*Phacelia argillacea*	—	—	—	—	—	—	—	—	—	—	E	—	—
Phacelia, North Park	*Phacelia formosula*	—	E	—	—	—	—	—	—	—	—	—	—	—
Twinpod, Dudley Bluffs	*Physaria obcordata*	—	PT	—	—	—	—	—	—	—	—	—	—	—
Orchid, White-fringed Prairie	*Platanthera leucophaea*	—	—	—	—	—	—	PT	PT	—	—	—	—	—
Orchid, Western Prairie Fringed	*Platanthera praeclara*	—	—	—	—	—	—	PT	PT	—	PT	—	—	—
Primrose, Maguire	*Primula maguirei*	—	—	—	—	—	—	—	—	—	—	T	—	—
Buttercup, Sharp Autumn	*Ranunculus acriformis* var. *aestivalis*	—	—	—	—	—	—	—	—	—	—	PE	—	—
Cactus, Unita Basin Hookless	*Sclerocactus glaucus* (=*Echinocactus g.*, *S. whipplei*)	—	T	—	—	—	—	—	—	—	—	T	—	—
Cactus, Mesa Verde	*Sclerocactus mesae - verdae* (=*Pediocactus m.*)	—	T	—	—	—	T	—	—	—	—	—	—	—
Cactus, Wright Fishhook	*Sclerocactus wrightiae* (=*Pediocactus w.*)	—	—	—	—	—	—	—	—	—	—	E	—	—
Groundsel, San Francisco Peaks	*Senecio franciscanus*	TCH	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Wire-lettuce, Malheur	*Stephanomeria malheurensis*	—	—	—	—	—	—	—	—	ECH	—	—	—	—
Townsendia, Last Chance	*Townsendia aprica*	—	—	—	—	—	—	—	—	—	—	T	—	—
Globe-berry, Tumamoc	*Tumamoca macdougalii*	E	—	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Mammals														
Bat, California Leaf-nosed	*Macrotus californicus*	P	—	—	—	—	P	—	—	—	—	—	—	—
Bat, Eastern Small-footed	*Myotis subulatus leibii*	—	—	—	—	—	—	—	P	—	—	—	—	—
Bat, Greater Western Mastiff	*Eumops perotis californicus*	P	—	—	—	—	P	—	—	—	—	—	—	—
Bat, Mexican Long-tongued	*Choenycteris mexicana*	P	—	—	—	—	P	—	—	—	—	—	—	—
Bat, Occult Little Brown	*Myotis lucifugus occultus*	P	—	—	—	—	P	—	—	—	—	—	—	—
Bat, Pacific Western Big-eared	*Plecotus townsendii townsendii*	—	—	P	—	—	—	—	—	P	—	—	P	—
Bat, Rafinesque's (=Southeastern) Big-eared	*Plecotus rafinesquii*	—	—	—	—	—	—	—	P	—	—	—	—	—
Bat, Southeastern Myotis	*Myotis austroriparius*	—	—	—	—	—	—	—	P	—	—	—	—	—
Bat, Southwestern Cave Myotis	*Myotis velifer brevis*	P	—	—	—	—	P	—	—	—	—	—	—	—
Bat, Spotted	*Euderma maculatum*	P	P	P	P	P	P	—	—	P	—	P	—	P
Bat, Underwood's Mastiff	*Eumops underwoodi*	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Chipmunk, Hidden Forest Uinta	*Eutamias umbrinus nevadensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Chipmunk, Mount Ellen Uinta	*Eutamias umbrinus sedulus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Chipmunk, Organ Mountains Colorado	*Eutamias quadrivittatus australis*	—	—	—	—	—	P	—	—	—	—	—	—	—
Chipmunk, Palmer's	*Eutamias palmeri*	—	—	—	—	P	—	—	—	—	—	—	—	—
Fox, Sierra Nevada Red	*Vulpes vulpes necator*	—	—	—	—	P	—	—	—	—	—	—	—	—
Fox, Swift	*Vulpes velox*	—	P	—	P	—	P	P	P	—	P	—	—	P
Gopher, Antelope Island Pocket	*Thomomys umbrinus nesophilus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, Bonneville Southern Pocket	*Thomomys umbrinus bonnevillei*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, Cebolleta Southern Pocket	*Thomomys umbrinus paguatae*	—	—	—	—	—	P	—	—	—	—	—	—	—
Gopher, Clear Lake Pocket	*Thomomys umbrinus convexus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, Fish Spring Pocket	*Thomomys umbrinus abstrusus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Gopher, Goldbeach Western Pocket	*Thomomys mazama helleri*	—	—	—	—	—	—	—	—	P	—	—	—	—
Gopher, Guadalupe Southern Pocket	*Thomomys umbrinus guadalupensis*	—	—	—	—	—	P	—	—	—	—	—	—	—
Gopher, Harquahala Pocket	*Thomomys umbrinus subsimilis*	P	—	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Gopher, Hualapai Southern Pocket	*Thomomys umbrinus hualpaiensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Gopher, Louie's Western Pocket	*Thomomys mazama louiei*	—	—	—	—	—	—	—	—	—	—	—	P	—
Gopher, Mearns' Southern Pocket	*Thomomys umbrinus mearnsi*	—	—	—	—	—	P	—	—	—	—	—	—	—
Gopher, Mount Ellen Pocket	*Thomomys umbrinus dissimilis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, Pajarito Southern Pocket	*Thomomys umbrinus quercinus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Gopher, Pistol River Pocket	*Thomomys umbrinus detumidus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Gopher, Prospect Valley Pocket	*Thomomys umbrinus muralis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Gopher, Roy Prairie Pocket	*Thomomys mazama glacialis*	—	—	—	—	—	—	—	—	—	—	—	P	—
Gopher, Salt Gulch Pocket	*Thomomys umbrinus powelli*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, San Antonio Pocket	*Thomomys umbrinus curtatus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Gopher, Searchlight Southern Pocket	*Thomomys umbrinus suboles*	P	—	—	—	—	—	—	—	—	—	—	—	—
Gopher, Skull Valley Pocket	*Thomomys umbrinus robustus*	—	—	—	—	—	—	—	—	—	—	P	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Gopher, Stansbury Island Pocket	*Thomomys umbrinus minimus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, Swasey Spring Pocket	*Thomomys umbrinus sevieri*	—	—	—	—	—	—	—	—	—	—	P	—	—
Gopher, Tacoma Western Pocket	*Thomomys mazama tacomensis*	—	—	—	—	—	—	—	—	—	—	—	P	—
Ground Squirrel, Allen's 13-lined	*Spermophilus tridecemlineatus alleni*	—	—	—	—	—	—	—	—	—	—	—	—	P
Ground Squirrel, Northern Idaho	*Spermophilus brunneus* ssp.	—	—	C	—	—	—	—	—	—	—	—	—	—
Ground Squirrel, Southern Idaho	*Spermophilus brunneus* ssp.	—	—	P	—	—	—	—	—	—	—	—	—	—
Kangaroo Mouse, Desert Valley	*Microdipodos megacephalus albiventer*	—	—	—	—	P	—	—	—	—	—	—	—	—
Kangaroo Mouse, Fletcher Dark	*Microdipodopos intermedius nasutus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Kangaroo Rat, Dolphin Island Chisel-toothed	*Dipodomys microps russeolus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Kangaroo Rat, Dolphin Island Awl-toothed	*Dipodomys ordii cineraceus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Kangaroo Rat, Gunnison Island	*Dipodomys microps alfredi*	—	—	—	—	—	—	—	—	—	—	P	—	—
Kangaroo Rat, Marble Canyon	*Dipodomys microps leucotis*	P	—	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Kangaroo Rat, Merriam's	*Dipodomys merriami frenatus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Kangaroo Rat, Texas	*Dipodomys elator*	—	—	—	—	—	—	—	P	—	—	—	—	—
Lynx, North American	*Felis lynx canadensis*	—	P	P	P	P	—	P	—	P	—	P	P	P
Marmot, Wet Mountains Yellow-bellied	*Marmota flaviventris notioros*	—	P	—	—	—	—	—	—	—	—	—	—	—
Mouse, Black Mountain Cactus	*Peromyscus eremicus pullus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Mouse, Black Mountain Pocket	*Perognathus intermedius nigrimontis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Mouse, Chiricahua Western Harvest	*Reithrodontomys megalotis arizonensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Mouse, Coconino Arizona Pocket	*Perognathus amplus ammodytes*	P	—	—	—	—	—	—	—	—	—	—	—	—
Mouse, New Mexican Jumping	*Zapus hudsonius luteus*	P	—	—	—	—	P	—	—	—	—	—	—	—
Mouse, Pinacate Cactus	*Peromyscus eremicus papagensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Mouse, Preble's Meadow Jumping	*Zapus hudsonius preblei*	—	P	—	—	—	—	—	—	—	—	—	—	P
Mouse, Silky Pocket	*Perognathus flavus goodpasteri*	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Mouse, Stansbury Island Harvest	*Reithrodontomys megalotis ravus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Mouse, Wupatki Arizona Pocket	*Perognathus amplus cineris*	P	—	—	—	—	—	—	—	—	—	—	—	—
Mouse, Yavapai Arizona Pocket	*Perognathus amplus amplus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Otter, Southwestern	*Lutra canadensis sonorae*	P	P	—	—	—	P	—	—	—	—	P	—	—
Pika, Barnes'	*Ochotona princeps barnesi*	—	—	—	—	—	—	—	—	—	—	P	—	—
Pika, Cinnamon	*Ochotona princeps cinnamomea*	—	—	—	—	—	—	—	—	—	—	P	—	—
Pika, Goat Peak	*Ochotona princeps nigrescens*	—	—	—	—	—	P	—	—	—	—	—	—	—
Pika, Heliotrope	*Ochotona princeps moorei*	—	—	—	—	—	—	—	—	—	—	P	—	—
Pika, Lasal	*Ochotona princeps lasalensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Pika, Wasatch	*Ochotona princeps wasatchensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Prairie Dog, Arizona Black-tailed	*Cynomys ludovicianus arizonensis*	P	—	—	—	—	P	—	—	—	—	—	—	—
Puma, Yuma	*Felis concolor browni*	P	—	—	—	—	—	—	—	—	—	—	—	—
Rabbit, White-sided Jack	*Lepus callotis gaillardi*	—	—	—	—	—	P	—	—	—	—	—	—	—
Rat, Hot Springs Cotton	*Sigmodon fulviventer goldmani*	—	—	—	—	—	P	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Rat, Yavapai Arizona Cotton	*Sigmodon arizonae jackson*	P	—	—	—	—	—	—	—	—	—	—	—	—
Rat, Yuma Hispid Cotton	*Sigmodon hispidus eremicus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Sheep, California Bighorn	*Ovis canadensis californiana*	—	—	—	—	—	—	—	—	P	—	—	P	—
Shrew, Arizona	*Sorex arizonae*	P	—	—	—	—	P	—	—	—	—	—	—	—
Shrew, Destruction Island	*Soret trowbridgii destructioni*	—	—	—	—	—	—	—	—	—	—	—	P	—
Shrew, Preble's	*Sorex preblei*	—	—	P	P	—	—	—	—	P	—	—	P	P
Skunk, Colorado Hog-nosed	*Conepatus mesoleucus figginsi*	—	P	—	—	—	—	—	—	—	—	—	—	—
Squirrel, Chiricahua Nayarit	*Sciurus nayaritensis chiricahuae*	P	—	—	—	—	—	—	—	—	—	—	—	—
Squirrel, Santa Catalina Mountains	*Sciurus arizonensis catalinae*	P	—	—	—	—	—	—	—	—	—	—	—	—
Vole, Ash Meadows Montane	*Microtus montanus nevadensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Vole, Navaho Mountain Mexican	*Microtus mexicanus navaho*	P	—	—	—	—	—	—	—	—	—	P	—	—
Vole, Pahranagat Valley Montane	*Microtus montanus fucosus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Vole, Potholes Meadow	*Microtus pennsylvanicus kincaidi*	—	—	—	—	—	—	—	—	—	—	—	P	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Vole, Shaw Island Townsend's	*Microtus townsendii pugeti*	—	—	—	—	—	—	—	—	—	—	—	P	—
Vole, Virgin River Montane	*Microtus montanus rivularis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Vole, White-footed	*Arborimus albipes*	—	—	—	—	—	—	—	—	P	—	—	—	—
Wolverine, California	*Gulo gulo luteus*	—	—	—	—	—	—	—	—	P	—	—	P	—
Wolverine, North American	*Gulo gulo luscus*	—	P	P	P	P	—	P	—	—	—	P	—	P
Woodrat, White Sands	*Neotoma micropus leucophaea*	—	—	—	—	—	P	—	—	—	—	—	—	—
Woodrat, Santa Catalina Mountains	*Neotoma mexicana bullata*	P	—	—	—	—	—	—	—	—	—	—	—	—
Birds														
Blackbird, Tricolored	*Agelaius tricolor*	—	—	—	—	—	—	—	—	P	—	—	—	—
Curlew, Long-billed	*Numenius americanus*	P	P	P	P	P	P	P	P	P	P	P	P	P
Duck, Fulvous Whistling	*Dendrocygna bicolor*	P	—	—	—	—	—	—	—	—	—	—	—	—
Flycatcher, Southwestern Willow	*Empidonax trailii extimus*	P	P	—	—	—	P	—	—	—	—	—	—	—
Goshawk, Apache Northern	*Accipiter gentilis apache*	P	—	—	—	—	P	—	—	—	—	—	—	—
Grouse, Columbian Sharptailed	*Tympanuchus phasianellus columbians*	—	P	P	P	P	—	—	—	P	—	P	P	P

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Grouse, Western Sage	*Centrocercus urophasianus phaios*	—	—	—	—	—	—	—	—	P	—	—	P	—
Hawk, Ferruginous	*Buteo regalis*	P	P	P	P	P	P	P	P	P	P	P	P	P
Hawk, Northern Gray	*Buteo nitidus maximus*	P	—	—	—	—	P	—	—	—	—	—	—	—
Ibis, White-faced	*Plegadis chihi*	P	P	P	—	P	P	—	P	P	—	P	—	P
Murrelet, Marbled	*Brachyramphus marmoratus*	—	—	—	—	—	—	—	—	P	—	—	P	—
Owl, Spotted	*Strix occidentalis*	P	P	—	—	—	P	—	—	P	—	P	P	—
Plover, Mountain	*Charadrius montanus*	P	P	—	P	P	P	P	P	—	P	P	—	P
Plover, Western Snowy	*Charadrius alexandrinus nivosus*	P	P	—	—	P	P	—	P	P	—	P	P	—
Pygmy-owl, Cactus Ferruginous	*Glaucidium brasiliarum cactorum*	P	—	—	—	—	—	—	—	—	—	—	—	—
Rail, California Black	*Laterallus jamaicensis coturniculus*	C	—	—	—	—	—	—	—	—	—	—	—	—
Shrike, Migrant Loggerhead	*Lanius ludovicianus migrans*	—	—	—	—	—	—	—	P	—	—	—	—	—
Sparrow, Bachman's	*Aimophila aestivalis*	—	—	—	—	—	—	—	P	—	—	—	—	—
Sparrow, Large-billed Savannah	*Passerculus sandwichensis rostratus*	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Fishes														
Chub, Alvord	*Gila alvordensis*	—	—	—	—	P	—	—	—	P	—	—	—	—
Chub, Big Smoky Valley Tui	*Gila bicolor* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Catlow Tui	*Gila bicolor* ssp.	—	—	—	—	—	—	—	—	P	—	—	—	—
Chub, Dixie Valley Tui	*Gila bicolor* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Fish Creek Springs Tui	*Gila bicolor* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Fish Lake Valley Tui	*Gila bicolor* spp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Gila	*Gila intermedia*	P	—	—	—	—	P	—	—	—	—	—	—	—
Chub, Gila Roundtail	*Gila robusta grahami*	P	—	—	—	—	P	—	—	—	—	—	—	—
Chub, Hot Creek Valley Tui	*Gila bicolor* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Lahontan Tui	*Gila bicolor obesa*	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Least	*Iotichthys phlegethontis*	—	—	—	—	—	—	—	—	—	—	C	—	—
Chub, Leatherside	*Gila copei*	—	—	P	—	—	—	—	—	—	—	P	—	P
Chub, Moapa Roundtail	*Gila robusta* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Newark Valley Tui	*Gila bicolor newarkensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Oregon	*Oregonichthys (=Hybopsis) crameri*	—	—	—	—	—	—	—	—	P	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Chub, Oregon Lakes Tui	*Gila bicolor*	—	—	—	—	—	—	—	—	P	—	—	—	—
Chub, Pleasant Valley Tui	*Gila bicolor* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Railroad Valley Tui	*Gila bicolor* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Chub, Sheldon Tui	*Gila bicolor eurysoma*	—	—	—	—	P	—	—	—	P	—	—	—	—
Chub, Sicklefin	*Hybopsis meeki*	—	—	—	—	—	—	P	—	—	P	—	—	—
Chub, Sturgeon	*Hybopsis gelida*	—	—	—	P	—	—	P	—	—	P	—	—	P
Chub, Summer Basin Tui	*Gila bicolor* ssp.	—	—	—	—	—	—	—	—	C	—	—	—	—
Dace, Diamond Valley Speckled	*Rhinichthys osculus* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Dace, Meadow Valley Wash Speckled	*Rhinichthys osculus* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Dace, Moapa Speckled	*Rhinichthys osculus moapae*	—	—	—	—	P	—	—	—	—	—	—	—	—
Dace, Monitor Valley Speckled	*Rhinichthys osculus* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Dace, Oasis Valley Speckled	*Rhinichthys osculus* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Dace, Pahranagat Speckled	*Rhinichthys osculus velifer*	—	—	—	—	P	—	—	—	—	—	—	—	—
Dace, Relict	*Relictus solitarius*	—	—	—	—	P	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Dace, White River Speckled	*Rhinichthys osculus* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Darter, Arkansas	*Etheostoma cragini*	—	P	—	—	—	—	—	P	—	—	—	—	—
Darter, Crystal	*Ammocrypta asprella*	—	—	—	—	—	—	—	P	—	—	—	—	—
Darter, Longnose	*Percina nasuta*	—	—	—	—	—	—	—	P	—	—	—	—	—
Grayling, Montana Arctic	*Thymallus arcticus montanus*	—	—	—	P	—	—	—	—	—	—	—	—	—
Madtom, Neosho	*Noturus placidus*	—	—	—	—	—	—	—	C	—	—	—	—	—
Mudminnow, Olympic	*Novumbra hubbsi*	—	—	—	—	—	—	—	—	—	—	—	P	—
Pupfish, Palomas	*Cyprinodon* sp.	—	—	—	—	—	P	—	—	—	—	—	—	—
Pupfish, Pecos	*Cyprinodon pecosensis*	—	—	—	—	—	C	—	—	—	—	—	—	—
Pupfish, White Sands	*Cyprinodon tularosa*	—	—	—	—	—	P	—	—	—	—	—	—	—
Sculpin, Malheur Mottled	*Cottus bairdi* ssp.	—	—	—	—	—	—	—	—	P	—	—	—	—
Sculpin, Slender	*Cottus tenuis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Sculpin, Wood River	*Cottus leiopomus*	—	—	P	—	—	—	—	—	—	—	—	—	—
Shiner, Arkansas River	*Notropis girardi*	—	—	—	—	—	P	—	P	—	—	—	—	—
Shiner, Ouachita Mountain	*Notropis snelsoni*	—	—	—	—	—	—	—	P	—	—	—	—	—
Shiner, Rio Grande	*Notropis jemezanus*	—	—	—	—	—	P	—	—	—	—	—	—	—
Spinedace, Virgin	*Lepidomeda mollispinis mollispinis*	P	—	—	—	P	—	—	—	—	—	P	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Springfish, Moapa White River	*Crenichthys baileyi moapa*	—	—	—	—	P	—	—	—	—	—	—	—	—
Springfish, Preston White River	*Crenichthys baileyi albivallis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Stoneroller, Mexican	*Campostoma ornatum*	P	—	—	—	—	—	—	—	—	—	—	—	—
Sturgeon, Lake	*Acipenser fulvescens*	—	—	—	—	—	—	—	—	—	P	—	—	—
Sturgeon, Pallid	*Scaphirhynchus albus*	—	—	—	C	—	—	C	—	C	—	—	—	—
Sucker, Blue	*Cycleptus elongatus*	—	—	—	P	—	P	P	P	—	P	—	—	—
Sucker, Goose Lake	*Catostomus occidentalis lacusanserinus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Sucker, Jenny Creek	*Catostomus rimiculus* ssp.	—	—	—	—	—	—	—	—	P	—	—	—	—
Sucker, Klamath Largescale	*Catostomus snyderi*	—	—	—	—	—	—	—	—	P	—	—	—	—
Sucker, Meadow Valley Wash Desert	*Catostomus clarkii* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Sucker, Razorback	*Xyrauchen texanus*	C	C	—	—	C	—	—	—	—	—	C	—	C
Sucker, Wall Canyon	*Catostomus* sp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Sucker, White River Desert	*Catostomus clarki intermedius*	—	—	—	—	P	—	—	—	—	—	—	—	—
Sucker, Zuni Mountain	*Catostomus discobolus yarrowi*	P	—	—	—	—	P	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Trout, Bonneville Cutthroat	*Salmo clarki utah*	—	—	P	—	P	—	—	—	—	—	P	—	P
Trout, Bull	*Salvelinus confluentus*	—	—	P	P	P	—	—	—	P	—	—	P	—
Trout, Colorado Cutthroat	*Salmo clarki pleuriticus*	—	P	—	—	—	—	—	—	—	—	P	—	P
Trout, Redband	*Salmo* sp.	—	—	P	—	P	—	—	—	P	—	—	—	—
Trout, Snake River Fine-spotted Cutthroat	*Salmo clarki* ssp.	—	—	P	—	—	—	—	—	—	—	—	—	—
Trout, Willow/ Whitehorse Cutthroat	*Salmo clarki* ssp.	—	—	—	—	—	—	—	—	P	—	—	—	—
Amphibians														
Frog, Tarahumara	*Rana tarahumarae*	C	—	—	—	—	—	—	—	—	—	—	—	—
Frog, Yavapai (=Lowland) Leopard	*Rana yavapaiensis*	P	—	—	—	—	P	—	—	—	—	—	—	—
Salamander, Del Norte	*Plethodon elongatus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Salamander, Jemez Mountain	*Plethodon neomexicanus*	—	—	—	—	—	C	—	—	—	—	—	—	—
Salamander, Larch Mountain	*Plethodon larselli*	—	—	—	—	—	—	—	—	P	—	—	P	—
Salamander, Oklahoma	*Eurycea tynerensis*	—	—	—	—	—	—	—	P	—	—	—	—	—
Salamander, Sacramento Mountains	*Aneides hardii*	—	—	—	—	—	P	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Salamander, Siskiyou Mountains	*Plethodon stormi* (=*P. elongatus stormi*)	—	—	—	—	—	—	—	—	P	—	—	—	—
Salamander, Sonoran Tiger	*Ambystoma tigrinum stebbinsi*	P	—	—	—	—	—	—	—	—	—	—	—	—
Toad, Amargosa	*Bufo nelsoni*	—	—	—	—	P	—	—	—	—	—	—	—	—
Toad, Arizona Southwestern	*Bufo microscaphus microscaphus*	P	—	—	—	P	P	—	—	—	—	P	—	—
Toad, Boreal Western (Rocky Mountains population)	*Bufo boreas boreas*	—	P	—	—	—	P	—	—	—	—	—	—	P
Reptiles														
Lizard, Cowles Fringe-toed	*Uma notata rufopunctata*	P	—	—	—	—	—	—	—	—	—	—	—	—
Lizard, Texas Horned	*Phrynosoma cornutum*	P	P	—	—	—	P	—	P	—	—	—	—	—
Lizard, Flat-tailed Horned	*Phrynosoma mcalli*	C	—	—	—	—	—	—	—	—	—	—	—	—
Skink, Arizona Gilbert's	*Eumeces gilberti arizonensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Snake, Mexican Garter	*Thamnophis eques*	P	—	—	—	—	P	—	—	—	—	—	—	—
Snake, Narrow-Headed Garter	*Thamnophis rufipunctatus*	P	—	—	—	—	P	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Tortoise, Desert (Mojave Desert population)	*Gopherus (=Xerobates) agassizii*	—	—	—	—	P	—	—	—	—	—	—	—	—
Tortoise, Desert Sonora (Sonora Desert population)	*Gopherus (=Xerobates) agassizii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Turtle, Alligator Snapping	*Macroclemys temmincki*	—	—	—	—	—	—	—	P	—	—	—	—	—
Turtle, Northwestern Pond	*Clemmys marmorata marmorata*	—	—	—	—	—	—	—	—	P	—	—	P	—
Whiptail, Gray-checkered Whiptail	*Cnemidophorus dixoni*	—	—	—	—	—	P	—	—	—	—	—	—	—

Insects

Beetle, Animas Minute Moss	*Limnebius aridus*	—	—	—	—	—	P	—	—	—	—	—	—	—
Beetle, Anthony Blister	*Lytta mirifica*	—	—	—	—	—	P	—	—	—	—	—	—	—
Beetle, Arizona Water Penny	*Psephenus arizonensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Beetle, Beer's False Water Penny	*Acneus beeri*	—	—	—	—	—	—	—	—	P	—	—	—	—
Beetle, Beller's Ground	*Agonum belleri*	—	—	—	—	—	—	—	—	P	—	—	P	—
Beetle, Big Dune Aphodius Scarab	*Ahodius* sp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Blind Cave Leiodid	*Glacicavicola bathysciodes*	—	—	P	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Beetle, Bonita Diving	*Deronectes neomexicana*	—	—	—	—	—	P	—	—	—	—	—	—	—
Beetle, Brown's Microcylloepus Riffle	*Microcylloepus browni*	—	—	—	P	—	—	—	—	—	—	—	—	—
Beetle, Burnell's False Water Penny	*Acneus burnelli*	—	—	—	—	—	—	—	—	P	—	—	—	—
Beetle, Chiricahua Water Scavenger	*Cymbiodyta arizonica*	P	—	—	—	—	—	—	—	—	—	—	—	—
Beetle, Coral Pink Dunes Tiger	*Cicindela limbata albissima*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beetle, Crescent Dune Aegialian Scarab	*Aegialia crescenta*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Crescent Dune Aphodius Scarab	*Aphodius* sp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Crescent Dune Serican Scarab	*Serica* sp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Death Valley Agabus Diving	*Agabus rumppi*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Devil's Hole Warm Spring Riffle	*Stenelmis calida calida*	—	—	—	—	P	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Beetle, Giuliani's Dune Scarab	*Pseudocotalpa giulianii*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Hardy's Aegialian Scarab	*Aegialia hardyi*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Hatch's Click	*Eanus hatchi*	—	—	—	—	—	—	—	—	—	—	—	P	—
Beetle, Idaho Dunes Tiger	*Cicindela arenicola*	—	—	P	—	—	—	—	—	—	—	—	—	—
Beetle, Large Aegialian Scarab	*Aegialia magnifica*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Little Riffle	*Dubiraphia parva*	—	—	—	—	—	—	—	P	—	—	—	—	—
Beetle, Los Olmos Tiger	*Cicindela nevadica olmosa*	—	—	—	—	—	P	—	—	—	—	—	—	—
Beetle, Maricopa Tiger	*Cicindela oregona maricopa*	P	—	—	—	—	—	—	—	—	—	—	—	—
Beetle, Marron's San Carlos Riffle	*Huleechius marroni carolus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Beetle, Moapa Warm Springs Riffle	*Stenelmis calida moapa*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Narrow-foot Hygrotus Diving	*Hygrotus diversipes*	—	—	—	—	—	—	—	—	—	—	—	—	P
Beetle, Parker's Riffle	*Cylloepus parkeri*	P	—	—	—	—	—	—	—	—	—	—	—	—
Beetle, Roth's Blind Ground	*Pterostichus rothi*	—	—	—	—	—	—	—	—	P	—	—	—	—

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Beetle, Sand Mountain Aphodius Scarab	*Aphodius* sp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Sand Mountain Serican Scarab	*Serica* sp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Beetle, Spangler's Hydroporus Diving	*Hydroporus spangleri*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beetle, Spotted Warner Valley Dunes June	*Polyphylla avittata*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beetle, Stephan's Riffle	*Heterelmis stephani*	P	—	—	—	—	—	—	—	—	—	—	—	—
Beetle, Utah Chaetarthrian Water Scavenger	*Chaetarthria utahensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beetle, Utah Hydroporus Diving	*Hydroporus utahensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beetle, Utah Minute Moss	*Limnebius utahensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beetle, Warm Spring Zaitzevian Riffle	*Zaitzevia thermae*	—	—	—	P	—	—	—	—	—	—	—	—	—
Beetle, White Mountains Water Penny	*Psephenus montanus*	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Bug, Amargosa Naucorid	*Pelocoris shoshone*	—	—	—	—	P	—	—	—	—	—	—	—	—
Bug, Santa Rita Mountains Chlorochroan	*Chlorochroa rita*	P	—	—	—	—	—	—	—	—	—	—	—	—
Bumblebee, Franklin's	*Bombus franklini*	—	—	—	—	—	—	—	—	P	—	—	—	—
Butterfly, Baking Powder Flat Blue	*Euphilotes battoides* ssp.	—	—	—	—	P	—	—	—	—	—	—	—	—
Butterfly, Blue Silverspot	*Speyeria nokomis caerulescens*	P	—	—	—	—	—	—	—	—	—	—	—	—
Butterfly, Carole's Silverspot	*Speyeria zerene carole*	—	—	—	—	P	—	—	—	—	—	—	—	—
Butterfly, Great Basin Silverspot	*Speyeria nokomis nokomis*	—	P	—	—	—	—	—	—	—	—	P	—	—
Butterfly, Mattoni's Blue	*Euphilotes (=Shijimaeoides) rita mattoni*	—	—	—	—	P	—	—	—	—	—	—	—	—
Butterfly, Mono Checkerspot	*Euphydryas editha monoensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Butterfly, Morand's Checkspot	*Euphydryas anicia morandi*	—	—	—	—	P	—	—	—	—	—	—	—	—
Butterfly, Regal Fritillary	*Speyeria idalia*	—	P	—	P	—	—	P	P	—	P	—	—	—
Butterfly, Spring Mountains Blue	*Plejebus shasta charlestonensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Butterfly, Tawny Crescent	*Phyciodes batesi*	—	—	—	—	—	—	P	—	—	P	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Butterfly, Uncompahgre Fritillary	*Boloria acrocnema*	—	C	—	—	—	—	—	—	—	—	—	—	—
Caddisfly, Abellan Hydropsyche	*Hydropsyche abella*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Alexander's Rhyacophilan	*Rhyacophila alexanderi*	—	—	—	P	—	—	—	—	—	—	—	—	—
Caddisfly, Alsea Ochrotrichian Micro	*Ochrotrichia alsea*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Blue Mountains Cryptochian	*Cryptochia neosa*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Cascades Apatanian	*Apatania (=Radema) tavala*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Clatsop Philocascan	*Philocasca oron*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Columbia Gorge Neothremman	*Neothremma andersoni*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Denning's Agapetus	*Agapetus denningi*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Deschutes Ochrotrichian Micro	*Ochrotrichia phenosa*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Fender's Rhyacophilan	*Rhyacophila fenderi*	—	—	—	—	—	—	—	—	P	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Caddisfly, Fort Dick Limnephilus	Limnephilus atercus	—	—	—	—	—	—	—	—	P				
Caddisfly, Goeden's Lepidostoman	Lepidostoma goedeni	—	—	—	—	—	—	—	—	P				
Caddisfly, Green Springs Mountain Farulan	Farula davisi	—	—	—	—	—	—	—	—	P				
Caddisfly, Haddock's Rhyacophilan	Rhyacophila haddocki	—	—	—	—	—	—	—	—	P				
Caddisfly, Mt. Hood Farulan	Farula jewetti	—	—	—	—	—	—	—	—	P				
Caddisfly, Mt. Hood Primitive Brachycentrid	Eobrachycentrus gelidae	—	—	—	—	—	—	—	—	P				
Caddisfly, Obrien Rhyacophilan	Rhyacophila colonus	—	—	—	—	—	—	—	—	P				
Caddisfly, One-spot Rhyacophilan	Rhyacophila unipunctata	—	—	—	—	—	—	—	—	P				
Caddisfly, Reisen's Hydropsyche	Hydropsyche reiseni	—	—	—	—	—	—	—	P	—				
Caddisfly, Schuh's Homoplectran	Homoplectra schuhi	—	—	—	—	—	—	—	—	P				
Caddisfly, Siskiyou	Tinodes siskiyou	—	—	—	—	—	—	—	—	P				
Caddisfly, Three-tooth Long-horned	Triaenodes tridonta	—	—	—	—	—	—	—	P	—				
Caddisfly, Tombstone Prairie Farulan	Farula reaperi	—	—	—	—	—	—	—	—	P	—	—	—	—

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Caddisfly, Tombstone Prairie Oligophlebodes	*Oligophlebodes mostbento*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Vertrees's Ceraclean	*Ceraclea (=Athripsodes) vertreesi*	—	—	—	—	—	—	—	—	P	—	—	—	—
Caddisfly, Vertrees's Ochrotrichian Micro	*Ochrotrichia vertreesi*	—	—	—	—	—	—	—	—	P	—	—	—	—
Cricket, Arizona Giant Sand Treader	*Daihinibaenetes arizonensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Cricket, Navajo Jerusalem	*Stenopelmatus navajo*	P	—	—	—	—	—	—	—	—	—	—	—	—
Cricket, Prairie Mole	*Gryllotalpa major*	—	—	—	—	—	—	—	P	—	—	—	—	—
Cricket, Tanner's Black Camel	*Utabaenetes tanneri*	—	—	—	—	—	—	—	—	—	—	P	—	—
Damselfly, Sabino Canyon	*Argia* sp.	P	—	—	—	—	—	—	—	—	—	—	—	—
Grasshopper, Big Cedar	*Eximacris phenax*	—	—	—	—	—	—	—	P	—	—	—	—	—
Grasshopper, Desert Monkey	*Psychomastix deserticola*	—	—	—	—	—	P	—	—	—	—	—	—	—
Grasshopper, Idaho Pointheaded	*Acrolophitus pulchellus*	—	—	P	—	—	—	—	—	—	—	—	—	—
Grasshopper, Pinaleno Monkey	*Eumorsea pinaleno*	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Grasshopper, Siskiyou Chloealtis	Chloealtis aspasma	—	—	—	—	—	—	—	—	P	—	—	—	—
Mayfly, Colorado Burrowing	Ephemera compar	—	P	—	—	—	—	—	—	—	—	—	—	—
Mayfly, False Ameletus	Ameletus falsus	P	—	—	—	—	—	—	—	—	—	—	—	—
(Millipede, no common name)	Toltecus chihuanus	—	—	—	—	—	P	—	—	—	—	—	—	—
Moth, Albarufan Dagger	Acronicta albarufa	—	P	—	—	—	P	—	—	—	—	—	—	—
Moth, Lost Ethmiid	Ethmia monachella	—	P	—	—	—	—	—	—	—	—	—	—	—
Moth, Stevens' Tortricid	Decodes stevensi	—	P	—	—	—	—	—	—	—	—	—	—	—
Owlfly, Cheese-weed	Oliarces clara	P	—	—	—	—	—	—	—	—	—	—	—	—
Skipper, Dakota	Hesperia dacotae	—	—	—	—	—	—	P	—	—	P	—	—	—
Skipper, MacNeill Sooty Wing	Hesperopsis gracielae	P	—	—	—	P	—	—	—	—	—	—	P	—
Skipper, Wandering	Pseudocopaeodes eunus eunus	P	—	—	—	P	—	—	—	—	—	—	—	—
Stonefly, Fender's Soliperlan	Soliperla fenderi	—	—	—	—	—	—	—	—	—	—	—	P	—
Stonefly, Lake Tahoe Benthic	Capnia lacustra	—	—	—	—	P	—	—	—	—	—	—	—	—
Stonefly, Meltwater Lednian	Lednia tumana	—	—	—	P	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Stonefly, Wahkeena Falls Flightless	*Nemoura wahkeena*	—	—	—	—	—	—	—	—	P	—	—	—	—
Wasp, Redheaded Sphecid	*Eucerceris ruficeps*	—	—	—	—	P	—	—	—	—	—	—	—	—
Weevil, Rulien's Miloderes	*Miloderes rulieni*	—	—	—	—	P	—	—	—	—	—	—	—	—
Crustaceans														
(Crayfish, no common name)	*Cambarus tartarus*	—	—	—	—	—	—	—	P	—	—	—	—	—
Amphipod, Arizona Cave	*Stygobromus (=Stygonectes) arizonensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Amphipod, Bowman's Cave	*Stygobromus (=Stygonectes) bowmani*	—	—	—	—	—	—	—	P	—	—	—	—	—
Amphipod, Malheur Cave	*Stygobromus hubbsi*	—	—	—	—	—	—	—	—	P	—	—	—	—
Amphipod, Noel's	*Gammarus desperatus*	—	—	—	—	—	P	—	—	—	—	—	—	—
Amphipod, Oklahoma Cave	*Allocrangonyx pellucidus*	—	—	—	—	—	—	—	P	—	—	—	—	—
Amphipod, Ozark Cave	*Stygobromus (=Stygonectes) ozarkensis*	—	—	—	—	—	—	—	P	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Isopod, Bat Cave	*Caecidotea macropoda*	—	—	—	—	—	—	—	P	—	—	—	—	—
Mollusks														
Fanshell, Western	*Cyprogenia aberti*	—	—	—	—	—	—	—	P	—	—	—	—	—
Hornshell, Texas (mussel)	*Popenaias popei*	—	—	—	—	—	P	—	—	—	—	—	—	—
Mountainsnail, Boulder Pile	*Oreohelix jugalis (=Oreohelox jugalis jugalis)*	—	—	P	—	—	—	—	—	—	—	—	—	—
Mountainsnail, Carinated Striate Banded	*Oreohelix strigosa goniogyra*	—	—	P	—	—	—	—	—	—	—	—	—	—
Mountainsnail, Coalville	*Oreohelix peripherica weberiana*	—	—	—	—	—	—	—	—	—	—	P	—	—
Mountainsnail, Idaho Banded	*Oreohelix idahoensis idahoensis*	—	—	P	—	—	—	—	—	—	—	—	—	—
Mountainsnail, Lava Rock (=Walton's Banded)	*Oreohelix waltoni*	—	—	P	—	—	—	—	—	—	—	—	—	—
Mountainsnail, Whorled	*Oreohelix vortex*	—	—	P	—	—	—	—	—	—	—	—	—	—
Mussel, Neosho Mucket (=Neosho Pearly Mussel)	*Lampsilis rafinesqueana*	—	—	—	—	—	—	—	P	—	—	—	—	—
Mussel (Scaleshell)	*Leptodea letodon*	—	—	—	—	—	—	—	P	—	—	—	—	—
Peaclam (No common name)	*Pisidium ultramontanum Prime*	—	—	—	—	—	—	—	—	P	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Peaclam, Sangre de Cristo	*Pisidium sanquinichristi*	—	—	—	—	—	P	—	—	—	—	—	—	—
Pebblesnail, Ash Meadows (=Point of Rocks Spring)	*Pyrgulopsis erythropoma*	—	—	—	—	C	—	—	—	—	—	—	—	—
Pebblesnail, Columbia (=Great Columbia River Spire Snail)	*Fluminicola (=Lithoglyphus) columbianus*	—	—	P	—	—	—	—	—	P	—	—	P	—
Pebblesnail, Moapa	*Fluminicola avernalis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Pebblesnail, Pahranagat	*Fluminicola merriami*	—	—	—	—	P	—	—	—	—	—	—	—	—
Pocketbook, Ouachita Rock	*Arkansia wheeleri*	—	—	—	—	—	—	—	C	—	—	—	—	—
Pondsnail, Thickshell (=Utah Band Snail)	*Stagicola utahensis (=Lymnaea Kingii)*	—	—	—	—	—	—	—	—	—	—	P	—	—
Slitmouth, Rich Mountain (=Pilsbry's Narrow-apertured Land Snail)	*Stenotrema pilsbryi*	—	—	—	—	—	—	—	C	—	—	—	—	—
(Snail, no common name)	*Monadenia fidelis minor*	—	—	—	—	—	—	—	—	P	—	—	—	—
(Snail, no common name)	*Valvata utahensis*	—	—	P	—	—	—	—	—	—	—	P	—	—
Snail, Amargosa Tryonia	*Tryonia variegata*	—	—	—	—	P	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Snail, Bliss Rapids	Genus and species undescribed	—	—	C	—	—	—	—	—	—	—	—	—	—
Snail, Cave Physa (=Wyoming Cave Snail)	Physella (=Physa) spelunca	—	—	—	—	—	—	—	—	—	—	—	—	P
Snail, Fish Lake Physa (=Fish Lake snail)	Physella (=Stenophysa) microstriata	—	—	—	—	—	—	—	—	—	—	P	—	—
Snail, Gila Tryonia	Tryonia gilae	P	—	—	—	—	—	—	—	—	—	—	—	—
Snail, Grated Tryonia (=White River snail)	Tryonia clathrata	—	—	—	—	P	—	—	—	—	—	—	—	—
Snail, Jackson Lake	Helisoma (Carinifex) jacksonense	—	—	—	—	—	—	—	—	—	—	—	—	P
Snail, Kanab amber	Oxylona haydeni kanabensis	—	—	—	—	—	—	—	—	—	—	P	—	—
Snail, Marbled Disc	Discus marmorensis	—	—	P	—	—	—	—	—	—	—	—	—	—
Snail, Minute Tryonia (=Minute Slender Tryonia Snail)	Tryonia ericae	—	—	—	—	C	—	—	—	—	—	—	—	—
Snail, Mission Creek Oregonian	Cryptomastix magnidentata	—	—	P	—	—	—	—	—	—	—	—	—	—
Snail, Newcomb's Littorine	Algamorda newcombiana (=Littorina subrotunda)	—	—	—	—	—	—	—	—	P	—	—	P	—
Snail, New Mexico Hotspring	'Fontelicella' thermalis	—	—	—	—	—	C	—	—	—	—	—	—	—
Snail, Pecos Assiminea	Assiminea pecos	—	—	—	—	—	C	—	—	—	—	—	—	—

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Snail, Point of Rocks Tryonia	*Tryonia elata*	—	—	—	—	C	—	—	—	—	—	—	—	—
Snail, Quitobaquito Tryonia	*Tryonia quitobaquitae*	P	—	—	—	—	—	—	—	—	—	—	—	—
Snail, San Xavier Talus	*Sonorella eremita*	P	—	—	—	—	—	—	—	—	—	—	—	—
Snail, Shortface Lanx	*Fisherola nuttallidorwa*	—	—	P	—	—	—	—	—	P	—	—	P	—
Snail, Snake River Physa	*Physa* sp.	—	—	C	—	—	—	—	—	—	—	—	—	—
Snail, Sportinggoods Tryonia	*Tryonia angulata*	—	—	—	—	C	—	—	—	—	—	—	—	—
Snail, Utah Physa	*Physella (=Physa) utahensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Snail, Virile Amargosa	Genus and species undescribed	—	—	—	—	C	—	—	—	—	—	—	—	—
Snail, Wet-rock Physa	*Physella (=Physa) zionis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Springsnail, Alamosa	*Tryonia alamosae*	—	—	—	—	—	C	—	—	—	—	—	—	—
Springsnail, Brown	*Pyrgulopsis solus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Bylas	*Apachecoccus arizonae*	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Chupadera	*'Fontelicella' chupaderae*	—	—	—	—	—	C	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Springsnail, Crystal Spring	*Pyrgulopsis cristalis*	—	—	—	—	C	—	—	—	—	—	—	—	—
Springsnail, Distal-Gland (=Large-gland Nevada Springnail)	*Pyrgulopsis nanus*	—	—	—	—	C	—	—	—	—	—	—	—	—
Springsnail, Elongate-gland	*Pyrgulopsis isolatus*	—	—	—	—	C	—	—	—	—	—	—	—	—
Springsnail, Fairbanks	*Pyrgulopsis fairbanksensis*	—	—	—	—	C	—	—	—	—	—	—	—	—
Springsnail, Fossil	*Pyrgulopsis simplex*	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Gila	*'Fontelicella' gilae*	—	—	—	—	—	C	—	—	—	—	—	—	—
Springsnail, Grand Wash	*Pyrgulopsis bacchus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Huachuca	*Pyrgulopsis thompsoni*	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Idaho	*Pyrgulopsis (=Fontelicella) idahoensis*	—	—	P	—	—	—	—	—	—	—	—	—	—
Springsnail, Jackson Lake (=Elk Island Snail)	*Pyrgulopsis (=Fontelicella) robusta*	—	—	—	—	—	—	—	—	—	—	—	—	P
Springsnail, Kingman	*Pyrgulopsis conicus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Koster's	*Tryonia kosteri*	—	—	—	—	—	P	—	—	—	—	—	—	—
Springsnail, Median-gland Nevada	*Pyrgulopsis pisteri*	—	—	—	—	C	—	—	—	—	—	—	—	—
Springsnail, Montezuma Well	*Pyrgulopsis montezumensis*	P	—	—	—	—	—	—	—	—	—	—	—	—

Part B: Candidate and Proposed Candidate Species

BLM Draft Vegetation Treatment EIS

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Springsnail, New Mexico Hot	'Fontelicella' thermalis	—	—	—	—	—	C	—	—	—	—	—	—	—
Springsnail, Oasis Valley	Pyrgulopsis (=Fontelicella) micrococcus	—	—	—	—	P	—	—	—	—	—	—	—	—
Springsnail, Page	Pyrgulopsis morrisoni	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Pecos	'Fontelicella' pecosensis	—	—	—	—	—	P	—	—	—	—	—	—	—
Springsnail, Roswell	'Fontelicella' roswellensis	—	—	—	—	—	C	—	—	—	—	—	—	—
Springsnail, San Bernadino	Yaquicoccus bernardinus	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Socorro	'Fontelicella' neomexicana	—	—	—	—	—	C	—	—	—	—	—	—	—
Springsnail, Three Forks	'Fontelicella' trivialis	P	—	—	—	—	—	—	—	—	—	—	—	—
Springsnail, Verde Rim	Pyrgulopsis glandulosus	P	—	—	—	—	—	—	—	—	—	—	—	—
Annelids														
Earthworm, Oregon Giant	Megascolides macelfreshi	—	—	—	—	—	—	—	—	P	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Turbellaria														
(Planarian, no common name)	*Kenkia rhynchida*	—	—	—	—	—	—	—	—	P	—	—	—	—
Arachnids														
Pseudoscorpion, Grand Canyon Cave	*Archeolarca cavicola*	P	—	—	—	—	—	—	—	—	—	—	—	—
Pseudoscorpion, Malheur	*Apochthonius malheuri*	—	—	—	—	—	—	—	—	P	—	—	—	—
Plants														
Sand-verbena, Rose-purple	*Abronia umbellata* ssp. *acutalata*	—	—	—	—	—	—	—	—	—	—	—	P	—
Abronia umbellata (Sci. Name)	*Abronia umbellata* ssp. *breviflora*	—	—	—	—	—	—	—	—	P	—	—	—	—
Foxglove, False	*Agalinis auriculata*	—	—	—	—	—	—	—	P	—	—	—	—	—
Agave parviflora (Sci. Name)	*Agave parviflora*	P	—	—	—	—	—	—	—	—	—	—	—	—
Agave schottii var. *treleasei* (Sci. Name)	*Agave schottii* var. *treleasei*	P	—	—	—	—	—	—	—	—	—	—	—	—
Bent Grass, Hendersons's	*Agrotis microphylla* var. *hendersonii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Bent Grass, Ross	*Agrostis rossiae*	—	—	—	—	—	—	—	—	—	—	—	—	P
Aletes humilis (Sci. Name)	*Aletes humilis*	—	P	—	—	—	—	—	—	—	—	—	—	—
Onion, Aase	*Allium aaseae*	—	—	C	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Onion, Blue Mountain	*Allium dictuon*	—	—	—	—	—	—	—	—	—	—	—	P	—
Allium douglasii var. *constrictum* (Sci. Name)	*Allium douglasii* var. *constrictum*	—	—	—	—	—	—	—	—	—	—	—	P	—
Allium gooddingii (Sci. Name)	*Allium gooddingii*	C	—	—	—	—	C	—	—	—	—	—	—	—
Ambrosia linearis (Sci. Name)	*Ambrosia linearis*	—	P	—	—	—	—	—	—	—	—	—	—	—
Indigo, False	*Amorpha ouachitensis*	—	—	—	—	—	—	—	C	—	—	—	—	—
Amsinckia carinata (Sci. Name)	*Amsinckia carinata*	—	—	—	—	—	—	—	—	P	—	—	—	—
Amsonia grandiflora (Sci. Name)	*Amsonia grandiflora*	P	—	—	—	—	—	—	—	—	—	—	—	—
Blue-star, Kearney's	*Amsonia kearneyana*	C	—	—	—	—	—	—	—	—	—	—	—	—
Amsonia peeblesii (Sci. Name)	*Amsonia peeblesii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Angelica scabrida (Sci. Name)	*Angelica scabrida*	—	—	—	—	C	—	—	—	—	—	—	—	—
Pussytoes, Meadow	*Antennaria arcuata*	—	—	P	—	P	—	—	—	—	—	—	—	P
Antennaria aromatica (Sci. Name)	*Antennaria aromatica*	—	—	—	—	—	—	—	—	—	—	—	—	P
Antennaria soliceps (Sci. Name)	*Antennaria soliceps*	—	—	—	—	C	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Dogbane, Jones'	*Apocynum jonesii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Columbine	*Aquilegia micrantha* var. *mancosana*	—	P	—	—	—	—	—	—	—	—	—	—	—
Rock Cress, Koehler's	*Arabis koehleri* var. *koehleri*	—	—	—	—	—	—	—	—	P	—	—	—	—
Arabis koehleri var. *stipitata* (Sci. Name)	*Arabis koehleri* var. *stipitata*	—	—	—	—	—	—	—	—	P	—	—	—	—
Arabis pusilla (Sci. Name)	*Arabis pusilla*	—	—	—	—	—	—	—	—	—	—	—	—	P
Arabis serpentinicola (Sci. Name)	*Arabis serpentinicola*	—	—	—	—	—	—	—	—	P	—	—	—	—
Rock Cress (Gray Knolls, Uintah Co.)	*Arabis* sp.	—	—	—	—	—	—	—	—	—	—	P	—	—
Arabis sp. (Del Norte, Curry Cos.) (Sci. Name)	*Arabis* sp. (Del Norte, Curry Cos.)	—	—	—	—	—	—	—	—	P	—	—	—	—
Arabis suffrutescens var. *horizontalis* (Sci. Name)	*Arabis suffrutescens* var. *horizontalis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Arabis williamsii (Sci. Name)	*Arabis williamsii*	—	—	—	—	—	—	—	—	—	—	—	—	P
Bear-poppy, Dwarf	*Arctomecon humilis*	—	—	—	—	—	—	—	—	—	—	C	—	—
Arenaria franklinii var. *thompsonii* (Sci. Name)	*Arenaria franklinii* var. *thompsonii*	—	—	—	—	—	—	—	—	P	—	—	P	—
Arenaria kingii ssp. *rosea* (Sci. Name)	*Arenaria kingii* ssp. *rosea*	—	—	—	—	C	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Arenaria paludicola (Sci. Name)	*Arenaria paludicola*	—	—	—	—	—	—	—	—	—	—	—	P	—
Argemone arizonica (Sci. Name)	*Argemone arizonica*	P	—	—	—	—	—	—	—	—	—	—	—	—
Arnica paniculata (Sci. Name)	*Arnica paniculata*	—	—	—	—	—	—	—	—	—	—	—	—	P
Sagebrush, Coaltown	*Artemisia argilosa*	—	P	—	—	—	—	—	—	—	—	—	—	—
Artemisia campestris var. *wormskioldii* (Sci. Name)	*Artemisia campestris* var. *wormskioldii*	—	—	—	—	—	—	—	—	P	—	—	P	—
Artemisia ludoviciana ssp. *estesii* (Sci. Name)	*Artemisia ludoviciana* ssp. *estesii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Milkweed, Cutler	*Asclepias cutleri*	P	—	—	—	—	—	—	—	—	—	P	—	—
Milkweed, Eastwood's	*Asclepias eastwoodiana*	—	—	—	—	P	—	—	—	—	—	—	—	—
Asplenium andrewsii (Sci. Name)	*Asplenium andrewsii*	P	P	—	—	—	—	—	—	—	—	P	—	—
Aster blepharophyllus (Sci. Name)	*Aster blepharophyllus*	—	—	—	—	—	P	—	—	—	—	—	—	—
Aster, Curtus	*Aster curtus*	—	—	—	—	—	—	—	—	P	—	—	P	—
Aster, Gorman	*Aster gormanii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Aster jessicae (Sci. Name)	*Aster jessicae*	—	—	P	—	—	—	—	—	—	—	—	P	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Aster lemmonii (Sci. Name)	*Aster lemmonii*	C	—	—	—	—	—	—	—	—	—	—	—	—
Aster mollis (Sci. Name)	*Aster mollis*	—	—	—	P	—	—	—	—	—	—	—	—	P
Aster vialis (Sci. Name)	*Aster vialis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Astragalus aequalis (Sci. Name)	*Astragalus aequalis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Milk-vetch, Gumbo	*Astragalus ampullarius*	P	—	—	—	—	—	—	—	—	—	P	—	—
Astragalus anserinus (Sci. Name)	*Astragalus anserinus*	—	—	—	—	P	—	—	—	—	—	P	—	—
Astragalus applegatei (Sci. Name)	*Astragalus applegatei*	—	—	—	—	—	—	—	—	P	—	—	—	—
Milk-vetch	*Astragalus atratus* var. *inseptus*	—	—	P	—	—	—	—	—	—	—	—	—	—
Astragalus barrii (Sci. Name)	*Astragalus barrii*	—	—	—	—	—	—	—	—	—	P	—	—	—
Milk-vetch, Beatley	*Astragalus beatleyae*	—	—	—	—	C	—	—	—	—	—	—	—	—
Astragalus camptopus (Sci. Name)	*Astragalus camptopus*	—	—	P	—	—	—	—	—	—	—	—	—	—
Astragalus collinus var. *laurentii* (Sci. Name)	*Astragalus collinus* var. *laurentii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Milk-vetch, Columbia	*Astragalus columbianus*	—	—	—	—	—	—	—	—	—	—	—	P	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Astragalus cottonii (Sci. Name)	*Astragalus cottonii*	—	—	—	—	—	—	—	—	—	—	—	P	—
Milk-vetch, Sentry	*Astragalus cremnophylax* var. *cremnophylax*)	C	—	—	—	—	—	—	—	—	—	—	—	—
Milk-vetch, Cronquist	*Astragalus cronquistii*	—	P	—	—	—	—	—	—	—	—	P	—	—
Astragalus debequaeus (Sci. Name)	*Astragalus debequaeus*	—	P	—	—	—	—	—	—	—	—	—	—	—
Milk-vetch, Deseret	*Astragalus desereticus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Astragalus diaphanus (Sci. Name)	*Astragalus diaphanus*	—	—	—	—	—	—	—	—	P	—	—	P	—
Milk-vetch	*Astragalus equisolensis*	—	—	—	—	—	—	—	—	—	—	C	—	—
Wooly Pod, Black	*Astragalus funereus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Astragalus geyeri var. *triquetrus* (Sci. Name)	*Astragalus geyeri* var. *triquetrus*	P	—	—	—	P	—	—	—	—	—	—	—	—
Milk-vetch, Gypsum	*Astragalus gypsodes*	—	—	—	—	—	P	—	—	—	—	—	—	—
Milk-vetch, Hamilton	*Astragalus hamiltonii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Milk-vetch, Harrison	*Astragalus harrisonii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Astragalus holmgreniorum (Sci. Name)	*Astragalus holmgreniorum*	P	—	—	—	—	—	—	—	—	—	P	—	—
Astragalus jejunus ssp. (Sci. Name)	*Astragalus jejunus* ssp.	—	—	—	—	—	—	—	—	—	—	—	—	P

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Milk-vetch, Douglas Thistle	Astragalus kentrophyta var. douglasii	—	—	—	—	—	—	—	—	P	—	—	P	—
Astragalus knightii (Sci. Name)	Astragalus knightii	—	—	—	—	—	P	—	—	—	—	—	—	—
Astragalus lentiginosus var. ambiguus (Sci. Name)	Astragalus lentiginosus var. ambiguus	P	—	—	—	—	—	—	—	—	—	—	—	—
Astragalus lentiginosus var. sesquimetralis (Sci. Name)	Astragalus lentiginosus var. sesquimetralis	—	—	—	—	C	—	—	—	—	—	—	—	—
Milk-vetch, Bear Valley	Astragalus lentiginosus var. ursinus	—	—	—	—	—	—	—	—	—	—	P	—	—
Milk-vetch, Grand Junction	Astragalus linifolius	—	P	—	—	—	—	—	—	—	—	P	—	—
Milk-vetch	Astragalus microcymbus	—	P	—	—	—	—	—	—	—	—	—	—	—
Milk-vetch, Pauper	Astragalus misellus var. pauper	—	—	—	—	—	—	—	—	—	—	—	P	—
Milk-vetch, Darwin Mesa	Astragalus mohavensis var. hemigyrus	—	—	—	—	P	—	—	—	—	—	—	—	—
Astragalus mulfordiae (Sci. Name)	Astragalus mulfordiae	—	—	P	—	—	—	—	—	P	—	—	—	—
Astragalus musimonum (Sci. Name)	Astragalus musimonum	P	—	—	—	P	—	—	—	—	—	—	—	—
Astragalus oniciformis (Sci. Name)	Astragalus oniciformis	—	—	P	—	—	—	—	—	—	—	—	—	—
Astragalus oophorus var. clokeyanus (Sci. Name)	Astragalus oophorus var. clokeyanus	—	—	—	—	C	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Milk-vetch, Osterhout	*Astragalus osterhoutii*	—	C	—	—	—	—	—	—	—	—	—	—	—
Astragalus peckii (Sci. Name)	*Astragalus peckii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Milk-vetch, Spring Mountain	*Astragalus remotus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Milk-vetch, Robbins	*Astragalus robbinsii* var. *alpiniformis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Milk-vetch, Robbins	*Astragalus robbinsii* var. *occidentalis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Astragalus sabulosus (Sci. Name)	*Astragalus sabulosus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Milk-vetch, Schmoll	*Astragalus schmolliae*	—	P	—	—	—	—	—	—	—	—	—	—	—
Astragalus shultziorum (Sci. Name)	*Astragalus shultziorum*	—	—	—	—	—	—	—	—	—	—	—	—	P
Milk-vetch, Whited	*Astragalus sinuatus*	—	—	—	—	—	—	—	—	—	—	—	P	—
Astragalus solitarius (Sci. Name)	*Astragalus solitarius*	—	—	—	—	P	—	—	—	P	—	—	—	—
Milk-vetch	*Astragalus sterilis*	—	—	P	—	—	—	—	—	P	—	—	—	—
Astragalus subcinereus var. *basalticus* (Sci. Name)	*Astragalus subcinereus* var. *basalticus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Astragalus tegetarioides (Sci. Name)	*Astragalus tegetarioides*	—	—	—	—	—	—	—	—	P	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Astragalus tephrodes var. eurylobus (Sci. Name)	*Astragalus tephrodes var. eurylobus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Astragalus titanophilus (Sci. Name)	*Astragalus titanophilus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Astragalus tyghensis (Sci. Name)	*Astragalus tyghensis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Milk-vetch	*Astragalus uncialis*	—	—	—	—	P	—	—	—	—	—	P	—	—
Astragalus vexilliflexus var. nubilus (Sci. Name)	*Astragalus vexilliflexus var. nubilus*	—	—	P	—	—	—	—	—	—	—	—	—	—
Milk-vetch, Gladiator	*Astragalus xiphoides*	C	—	—	—	—	—	—	—	—	—	—	—	—
Milk-vetch, Osgood Mountains	*Astragalus yoder-wiliamsii*	—	—	P	—	P	—	—	—	—	—	—	—	—
Atriplex canescens var. gigantea (Sci. Name)	*Atriplex canescens var. gigantea*	—	—	—	—	—	—	—	—	—	—	P	—	—
Saltbush	*Atriplex pleiantha*	—	P	—	—	—	P	—	—	—	—	P	—	—
Balsamorhiza sericea (Sci. Name)	*Balsamorhiza sericea*	—	—	—	—	—	—	—	—	P	—	—	—	—
Bensoniella oregona (Sci. Name)	*Bensoniella oregona*	—	—	—	—	—	—	—	—	P	—	—	—	—
Botrychium paradoxum (Sci. Name)	*Botrychium paradoxum*	—	—	—	P	—	—	—	—	—	—	—	—	—
Grape Fern, Crater Lake	*Botrychium pumicola*	—	—	—	—	—	—	—	—	P	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Braya humilis ssp. *ventosa* (Sci. Name)	*Braya humilis* ssp. *ventosa*	—	P	—	—	—	—	—	—	—	—	—	—	—
Reed Grass, Thurber's	*Calamagrostis crassiglumis*	—	—	—	—	—	—	—	—	—	—	—	P	—
Reed Grass	*Calamagrostis tweedyi*	—	—	P	P	—	—	—	—	—	—	—	P	—
Sand Grass	*Calamovilfa arcuata*	—	—	—	—	—	—	—	C	—	—	—	—	—
Poppy-mallow	*Callirhoe bushii*	—	—	—	—	—	—	—	P	—	—	—	—	—
Mariposa, Greene's	*Calochortus greenei*	—	—	—	—	—	—	—	—	P	—	—	—	—
Calochortus howellii (Sci. Name)	*Calochortus howellii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Mariposa	*Calochortus indecorus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Star-tulip, Long-haired	*Calochortus longebarbatus* var. *longebarbatus*	—	—	—	—	—	—	—	—	P	—	—	P	—
Mariposa-lily, Peck's Long-bearded	*Calochortus longebarbatus* var. *peckii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Calochortus nitidus (Sci. Name)	*Calochortus nitidus*	—	—	P	—	—	—	—	—	—	—	—	P	—
Mariposa, Alkali	*Calochortus striatus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Camissonia confertiflora (Sci. Name)	*Camissonia confertiflora*	P	—	—	—	—	—	—	—	—	—	—	—	—
Camissonia exilis (Sci. Name)	*Camissonia exilis*	P	—	—	—	—	—	—	—	—	—	P	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Camissonia megalantha (Sci. Name)	*Camissonia megalantha*	—	—	—	—	P	—	—	—	—	—	—	—	—
Camissonia specuicola ssp. *hesperia* (Sci. Name)	*Camissonia specuicola* ssp. *hesperia*	P	—	—	—	—	—	—	—	—	—	—	—	—
Bitter Cress, Saddle Mountain	*Cardamine pattersonii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Sedge, Indian Valley	*Carex aboriginum*	—	—	P	—	—	—	—	—	—	—	—	—	—
Carex fissa (Sci. Name)	*Carex fissa*	—	—	—	—	—	—	—	P	—	—	—	—	—
Sedge, Waterfall's	*Carex latebracteata*	—	—	—	—	—	—	—	C	—	—	—	—	—
Carex lenticularis var. *dolia* (Sci. Name)	*Carex lenticularis* var. *dolia*	—	—	—	P	—	—	—	—	—	—	—	—	—
Chinquapin, Ozark	*Castanea pumila* var. *ozarkensis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Indian Paintbrush, Aquarius	*Castilleja aquariensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Indian Paintbrush, Green-tinged	*Castilleja chlorotica*	—	—	—	—	—	—	—	—	P	—	—	—	—
Indian Paintbrush, Christ's	*Castilleja christii*	—	—	C	—	—	—	—	—	—	—	—	—	—
Castilleja cryptantha (Sci. Name)	*Castilleja cryptantha*	—	—	—	—	—	—	—	—	—	—	—	P	—
Castilleja fraterna (Sci. Name)	*Castilleja fraterna*	—	—	—	—	—	—	—	—	P	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Castilleja kaibabensis (Sci. Name)	Castilleja kaibabensis	P	—	—	—	—	—	—	—	—	—	—	—	—
Castilleja levisecta (Sci. Name)	Castilleja levisecta	—	—	—	—	—	—	—	—	P	—	—	P	—
Indian Paintbrush, Reveal	Castilleja revealii	—	—	—	—	—	—	—	—	—	—	P	—	—
Indian Paintbrush	Castilleja salsuginosa	—	—	—	—	P	—	—	—	—	—	—	—	—
Castilleja steenensis (Sci. Name)	Castilleja steenensis	—	—	—	—	—	—	—	—	P	—	—	—	—
Castilleja xanthotricha (Sci. Name)	Castilleja xanthotricha	—	—	—	—	—	—	—	—	P	—	—	—	—
Cereus greggii (Sci. Name)	Cereus greggii	P	—	—	—	—	P	—	—	—	—	—	—	—
Chaetopappa elegans (Sci. Name)	Chaetopappa elegans	—	—	—	—	—	P	—	—	—	—	—	—	—
Chaetopappa hersheyi (Sci. Name)	Chaetopappa hersheyi	—	—	—	—	—	P	—	—	—	—	—	—	—
Cheilanthes arizonica (Sci. Name)	Cheilanthes arizonica	P	—	—	—	—	—	—	—	—	—	—	—	—
Cheilanthes pringlei (Sci. Name)	Cheilanthes pringlei	P	—	—	—	—	—	—	—	—	—	—	—	—
Choisya mollis (Sci. Name)	Choisya mollis	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Chrysothamnus molestus (Sci. Name)	*Chrysothamnus molestus*	P	—	—	—	—	—	—	—	—	—	—	—	—
Cimicifuga arizonica (Sci. Name)	*Cimicifuga arizonica*	C	—	—	—	—	—	—	—	—	—	—	—	—
Thistle, Ashland	*Cirsium ciliolatum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Thistle, Ownbey's	*Cirsium ownbeyi*	—	—	—	—	—	—	—	—	—	—	P	—	—
Cirsium virginensis (Sci. Name)	*Cirsium virginensis*	P	—	—	—	—	—	—	—	—	—	P	—	—
Claytonia lanceolata var. *flava* (Sci. Name)	*Claytonia lanceolata* var. *flava*	—	—	P	P	—	—	—	—	—	—	—	—	—
Clematis hirsutissima var. *arizonica* (Sci. Name)	*Clematis hirsutissima* var. *arizonica*	P	—	—	—	—	—	—	—	—	—	—	—	—
Cleome multicaulis (Sci. Name)	*Cleome multicaulis*	P	P	—	—	—	P	—	—	—	—	—	—	P
Collomia, Bristle-flowered	*Collomia macrocalyx*	—	—	—	—	—	—	—	—	P	—	—	—	—
Collomia mazama (Sci. Name)	*Collomia mazama*	—	—	—	—	—	—	—	—	P	—	—	—	—
Bird's-beak, North Coast	*Cordylanthus maritimus* ssp. *palustris*	—	—	—	—	—	—	—	—	P	—	—	—	—
Bird's-beak, Tecopa	*Cordylanthus tecopensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Corydalis aquae-gelidae (Sci. Name)	*Corydalis aquae-gelidae*	—	—	—	—	—	—	—	—	P	—	—	P	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Coryphantha missouriensis var. *marstonii* (Sci. Name)	*Coryphantha missouriensis* var. *marstonii*	P	—	—	—	—	—	—	—	—	—	P	—	—
Coryphantha recurvata (Sci. Name)	*Coryphantha recurvata*	P	—	—	—	—	—	—	—	—	—	—	—	—
Coryphantha scheeri var. *robustispina* (Sci. Name)	*Coryphantha scheeri* var. *robustispina*	C	—	—	—	—	—	—	—	—	—	—	—	—
Catseye	*Cryptantha aperta*	—	P	—	—	—	—	—	—	—	—	—	—	—
Catseye, Barneby	*Cryptantha barnebyi*	—	—	—	—	—	—	—	—	—	—	C	—	—
Catseye, Compact	*Cryptantha compacta*	—	—	—	—	—	—	—	—	—	—	P	—	—
Cryptantha creutzfeldtii (Sci. Name)	*Cryptantha creutzfeldtii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Cryptantha hoffmannii (Sci. Name)	*Cryptantha hoffmannii*	—	—	—	—	P	—	—	—	—	—	—	—	—
Catseye	*Cryptantha insolita*	—	—	—	—	P	—	—	—	—	—	—	—	—
Catseye, Yellow-white	*Cryptantha ochroleuca*	—	—	—	—	—	—	—	—	—	—	P	—	—
Cryptantha subcapitata (Sci. Name)	*Cryptantha subcapitata*	—	—	—	—	—	—	—	—	—	—	—	—	P
Dodder	*Cuscuta attenuata*	—	—	—	—	—	—	—	C	—	—	—	—	—
Dodder, Warner's	*Cuscuta warneri*	—	—	—	—	—	—	—	—	—	—	P	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Cymopterus beckii (Sci. Name)	*Cymopterus beckii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Cymopterus goodrichii (Sci. Name)	*Cymopterus goodrichii*	—	—	—	—	P	—	—	—	—	—	—	—	—
Biscuitroot, Higgins	*Cymopterus higginsii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Biscuitroot, Cedar Breaks	*Cymopterus minimus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Cymopterus nivalis (Sci. Name)	*Cymopterus nivalis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Cymopterus ripleyi var. *saniculoides* (Sci. Name)	*Cymopterus ripleyi* var. *saniculoides*	—	—	—	—	P	—	—	—	—	—	—	—	—
Cymopterus sp. (Sci. Name)	*Cymopterus* sp.	—	—	—	—	—	—	—	—	—	—	—	—	P
Cymopterus sp. (Sci. Name)	*Cymopterus* sp.	—	—	P	—	—	—	—	—	—	—	—	—	—
Cynanchum wigginsi (Sci. Name)	*Cynanchum wigginsii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Prairie-Clover, Hole-in-the-Rock	*Dalea epica*	—	—	—	—	—	—	—	—	—	—	P	—	—
Indigobush, Gentry's	*Dalea tentaculoides*	C	—	—	—	—	—	—	—	—	—	—	—	—
Delphinium leucophaeum (Sci. Name)	*Delphinium leucophaeum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Delphinium pavonaceum (Sci. Name)	*Delphinium pavonaceum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Larkspur, Wenatchee	*Delphinium viridescens*	—	—	—	—	—	—	—	—	—	—	—	C	—
Larkspur	*Descurainia torulosa*	—	—	—	—	—	—	—	—	—	—	—	—	P

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Douglasia idahoensis (Sci. Name)	Douglasia idahoensis	—	—	P	—	—	—	—	—	—	—	—	—	—
Draba aprica (Sci. Name)	Draba aprica	—	—	—	—	—	—	—	P	—	—	—	—	—
Draba arida (Sci. Name)	Draba arida	—	—	—	—	P	—	—	—	—	—	—	—	—
Draba jaegeri (Sci. Name)	Draba jaegeri	—	—	—	—	P	—	—	—	—	—	—	—	—
Draba lemmonii var. cyclomorpha (Sci. Name)	Draba lemmonii var. cyclomorpha	—	—	—	—	—	—	—	—	P	—	—	—	—
Draba maguirei var. burkei (Sci. Name)	Draba maguirei var. burkei	—	—	—	—	—	—	—	—	—	—	P	—	—
Draba paucifructa (Sci. Name)	Draba paucifructa	—	—	—	—	P	—	—	—	—	—	—	—	—
Waterweed, Nevada	Elodea nevadensis	—	—	—	—	P	—	—	—	—	—	—	—	—
Epilobium oreganum (Sci. Name)	Epilobium oreganum	—	—	—	—	—	—	—	—	P	—	—	—	—
Daisy, Basalt	Erigeron basalticus	—	—	—	—	—	—	—	—	—	—	—	C	—
Erigeron chrysopsidis var. brevifolius (Sci. Name)	Erigeron chrysopsidis var. brevifolius	—	—	—	—	—	—	—	—	P	—	—	—	—
Daisy, Cronquist	Erigeron cronquistii	—	—	—	—	—	—	—	—	—	—	P	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Erigeron decumbens var. decumbens (Sci. Name)	*Erigeron decumbens var. decumbens*	—	—	—	—	—	—	—	—	P	—	—	—	—
Erigeron hessi (Sci. Name)	*Erigeron hessii*	—	—	—	—	—	P	—	—	—	—	—	—	—
Fleabane, Howell's	*Erigeron howellii*	—	—	—	—	—	—	—	—	P	—	—	P	—
Daisy, Kachina	*Erigeron kachinensis*	—	P	—	—	—	—	—	—	—	—	P	—	—
Fleabane	*Erigeron kuschei*	C	—	—	—	—	—	—	—	—	—	—	—	—
Fleabane	*Erigeron latus*	—	—	P	—	P	—	—	—	—	—	—	—	—
Erigeron lemmonii (Sci. Name)	*Erigeron lemmonii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Erigeron maguirei var. harrisonii (Sci. Name)	*Erigeron maguirei var. harrisonii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Daisy, Depauperate	*Erigeron mancus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Erigeron ovinus (Sci. Name)	*Erigeron ovinus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Erigeron pringlei (Sci. Name)	*Erigeron pringlei*	P	—	—	—	—	—	—	—	—	—	—	—	—
Erigeron sionis (Sci. Name)	*Erigeron sionis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Erigeron sp. (Sci. Name)	*Erigeron* sp.	—	—	—	—	—	—	—	—	—	—	—	—	P
Erigeron untermannii (Sci. Name)	*Erigeron untermannii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Pipewort	*Eriocaulon kornickianum*	—	—	—	—	—	—	—	P	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Wild-buckwheat, Sand-loving	*Eriogonum ammophilum*	—	—	—	—	—	—	—	—	—	—	C	—	—
Eriogonum apachense (Sci. Name)	*Eriogonum apachense*	P	—	—	—	—	—	—	—	—	—	—	—	—
Wild-buckwheat, Widstoe	*Eriogonum aretioides*	—	—	—	—	—	—	—	—	—	—	P	—	—
Wild-buckwheat	*Eriogonum argophyllum*	—	—	—	—	P	—	—	—	—	—	—	—	—
Eriogonum bifurcatum (Sci. Name)	*Eriogonum bifurcatum*	—	—	—	—	P	—	—	—	—	—	—	—	—
Wild-buckwheat, Brandegee	*Eriogonum brandegei*	—	P	—	—	—	—	—	—	—	—	—	—	—
Wild-buckwheat	*Eriogonum capillare*	P	—	—	—	—	—	—	—	—	—	—	—	—
Wild-buckwheat, Golden	*Eriogonum chrysops*	—	—	—	—	—	—	—	—	P	—	—	—	—
Wild-buckwheat, Cronquist	*Eriogonum cronquistii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Eriogonum crosbyae (Sci. Name)	*Eriogonum crosbyae*	—	—	—	—	—	—	—	—	P	—	—	—	—
Eriogonum cusickii (Sci. Name)	*Eriogonum cusickii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Eriogonum holmgrenii (Sci. Name)	*Eriogonum holmgrenii*	—	—	—	—	P	—	—	—	—	—	—	—	—
Eriogonum lagopus (Sci. Name)	*Eriogonum lagopus*	—	—	—	P	—	—	—	—	—	—	—	—	P

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Eriogonum lobbii var. *robustum* (Sci. Name)	*Eriogonum lobbii* var. *robustum*	—	—	—	—	P	—	—	—	—	—	—	—	—
Wild-buckwheat	*Eriogonum mortonianum*	P	—	—	—	—	—	—	—	—	—	—	—	—
Wild-buckwheat, Prostrate	*Eriogonum prociduum*	—	—	—	—	P	—	—	—	P	—	—	—	—
Eriogonum ripleyi (Sci. Name)	*Eriogonum ripleyi*	P	—	—	—	—	—	—	—	—	—	—	—	—
Eriogonum scopulorum (Sci. Name)	*Eriogonum scopulorum*	—	—	—	—	—	—	—	—	—	P	—	—	—
Wild-buckwheat, Smith	*Eriogonum smithii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Eriogonum soredium (Sci. Name)	*Eriogonum soredium*	—	—	—	—	—	—	—	—	—	—	Pʲ	—	—
Wild-buckwheat Thompson Atwood's	*Eriogonum thompsonae* var. *atwoodii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Wild-buckwheat	*Eriogonum viscidulum*	—	—	—	—	P	—	—	—	—	—	—	—	—
Eriogonum visheri (Sci. Name)	*Eriogonum visheri*	—	—	—	—	—	—	—	—	—	P	—	—	P
Erythronium sp. (Sci. Name)	*Erythronium* sp.	—	—	—	—	—	—	—	—	—	P	—	—	—
Spurge, Flat-seeded	*Euphorbia platysperma*	P	—	—	—	—	—	—	—	—	—	—	—	—
Ferocactus acanthodes var. *acanthodes* (Sci. Name)	*Ferocactus acanthodes* var. *acanthodes*	P	—	—	—	—	—	—	—	—	—	—	—	—
Fescue, Sedge	*Festuca dasyclada*	—	P	—	—	—	—	—	—	—	—	P	—	—

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Festuca hallii (Sci. Name)	*Festuca hallii*	—	P	—	—	—	—	—	—	—	—	—	—	—
Flaveria macdougallii (Sci. Name)	*Flaveria macdougallii*	C	—	—	—	—	—	—	—	—	—	—	—	—
Frasera coloradensis (Sci. Name)	*Frasera coloradensis*	—	P	—	—	—	—	—	—	—	—	—	—	—
Green-gentian	*Frasera gypsicola*	—	—	—	—	C	—	—	—	—	—	C	—	—
Green-gentian	*Frasera pahutensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Green-gentian, Umpqua	*Frasera umpquaensis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Mission-bells, Gentner	*Fritillaria gentneri*	—	—	—	—	—	—	—	—	P	—	—	—	—
Blanketflower, Yellow	*Gaillardia flava*	—	—	—	—	—	—	—	—	—	—	P	—	—
Bedstraw, Kingston	*Galium hilendiae* ssp. *kingstonense*	—	—	—	—	P	—	—	—	—	—	—	—	—
Gaura neomexicana ssp. *coloradensis* (Sci. Name)	*Gaura neomexicana* ssp. *coloradensis*	—	C	—	—	—	—	—	—	—	—	—	—	C
Gentian	*Gentiana bisetaea*	—	—	—	—	—	—	—	—	P	—	—	—	—
Gilia, Rabbit Valley	*Gilia caespitosa*	—	—	—	—	—	—	—	—	—	—	C	—	—
Gilia, Beautiful	*Gilia formosa*	—	—	—	—	—	P	—	—	—	—	—	—	—
Graptopetalum bartramii (Sci. Name)	*Graptopetalum bartramii*	P	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Hedge-hyssop, Boggs Lake	*Gratiola heterosepala*	—	—	—	—	—	—	—	—	P	—	—	—	—
Grindelia howellii (Sci. Name)	*Grindelia howellii*	—	—	P	P	—	—	—	—	—	—	—	—	—
Stickseed, Cronquist's	*Hackelia cronquistii*	—	—	—	—	—	—	—	—	C	—	—	—	—
Stickseed	*Hackelia ibapensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Stickseed, Showy	*Hackelia venusta*	—	—	—	—	—	—	—	—	—	—	—	P	—
Halimolobos perplexa var. *perplexa* (Sci. Name)	*Halimolobos perplexa* var. *perplexa*	—	—	P	—	—	—	—	—	—	—	—	—	—
Haplopappus alpinus (Sci. Name)	*Haplopappus alpinus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Goldenweed	*Haplopappus fremontii* ssp. *monocephalus*	—	P	—	—	—	—	—	—	—	—	—	—	—
Haplopappus insecticruris (Sci. Name)	*Haplopappus insecticruris*	—	—	P	—	—	—	—	—	—	—	—	—	—
Haplopappus liatriformis (Sci. Name)	*Haplopappus liatriformis*	—	—	P	—	—	—	—	—	—	—	—	P	—
Goldenweed	*Haplopappus radiatus*	—	—	P	—	—	—	—	—	P	—	—	—	—
Hastingsia bracteosa (Sci. Name)	*Hastingsia bracteosa*	—	—	—	—	—	—	—	—	C	—	—	—	—
Hedysarum occidentale var. *canone* (Sci. Name)	*Hedysarum occidentale* var. *canone*	—	—	—	—	—	—	—	—	—	—	P	—	—
Sunflower	*Helianthus paradoxus*	—	—	—	—	—	C	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Golden-aster, Jones	*Heterotheca jonesii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Horkelia hendersonii (Sci. Name)	*Horkelia hendersonii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Howellia aquatilis (Sci. Name)	*Howellia aquatilis*	—	—	P	P	—	—	—	—	P	—	—	P	—
Hymenoxys depressa (Sci. Name)	*Hymenoxys depressa*	—	—	—	—	—	—	—	—	—	—	P	—	—
Hymenoxys helenioides (Sci. Name)	*Hymenoxys helenioides*	P	P	—	—	—	—	—	—	—	—	P	—	—
Morning-glory, Lemmon's	*Ipomoea lemmonii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Ipomopsis polyantha (Sci. Name)	*Ipomopsis polyantha*	—	P	—	—	—	—	—	—	—	—	—	—	—
Ipomopsis polyantha var. *polyantha* (Sci. Name)	*Ipomopsis polyantha* var. *polyantha*	—	P	—	—	—	—	—	—	—	—	—	—	—
Ivesia cryptocaulis (Sci. Name)	*Ivesia cryptocaulis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Ivesia, Grimy	*Ivesia rhypara*	—	—	—	—	P	—	—	—	P	—	—	—	—
Lasthenia macrantha ssp. *prisca* (Sci. Name)	*Lasthenia macrantha* ssp. *prisca*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lathyrus holochlorus (Sci. Name)	*Lathyrus holochlorus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Pepper Cress, Barneby	*Lepidium barnebyanum*	—	—	—	—	—	—	—	—	—	—	C	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Pepper Cress, Davis'	*Lepidium davisii*	—	—	P	—	—	—	—	—	P	—	—	—	—
Lepidium montanum var. *neeseae* (Sci. Name)	*Lepidium montanum* var. *neeseae*	—	—	—	—	—	—	—	—	—	—	P	—	—
Lepidium montanum var. *stellae* (Sci. Name)	*Lepidium montanum* var. *stellae*	—	—	—	—	—	—	—	—	—	—	C	—	—
Lepidium ostleri (Sci. Name)	*Lepidium ostleri*	—	—	—	—	—	—	—	—	—	—	P	—	—
Lepidospartum burgessii (Sci. Name)	*Lepidospartum burgessii*	—	—	—	—	—	P	—	—	—	—	—	—	—
Lesquerella condensata (Sci. Name)	*Lesquerella condensata*	—	P	—	—	—	—	—	—	—	—	—	—	—
Lesquerella kaibabensis (Sci. Name)	*Lesquerella kaibabensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Bladder-pod	*Lesquerella parviflora*	—	P	—	—	—	—	—	—	—	—	—	—	—
Bladder-pod	*Lesquerella pruinosa*	—	P	—	—	—	—	—	—	—	—	—	—	—
Bladder-pod	*Lesquerella tumulosa*	—	—	—	—	—	—	—	—	—	—	P	—	—
Lewisia, Howell's	*Lewisia cotyledon* var. *howellii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lewisia cotyledon var. *purdyi* (Sci. Name)	*Lewisia cotyledon* var. *purdyi*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lewisia maguirei (Sci. Name)	*Lewisia maguirei*	—	—	—	—	P	—	—	—	—	—	—	—	—
Lilaeopsis recurva (Sci. Name)	*Lilaeopsis recurva*	P	—	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Lily, Western	*Lilium occidentale*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lilium parryi (Sci. Name)	*Lilium parryi*	P	—	—	—	—	—	—	—	—	—	—	—	—
Meadowfoam, Bellinger's	*Limnanthes floccosa* ssp. *bellingerana*	—	—	—	—	—	—	—	—	P	—	—	—	—
Meadowfoam, Wooly, Large-flowered	*Limnanthes floccosa* ssp. *grandiflora*	—	—	—	—	—	—	—	—	P	—	—	—	—
Meadowfoam, Wooly Dwarf	*Limnanthes floccosa* ssp. *pumila*	—	—	—	—	—	—	—	—	C	—	—	—	—
Limnanthes gracilis var. *gracilis* (Sci. Name)	*Limnanthes gracilis* var. *gracilis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Mudwort	*Limosella pubiflora*	P	—	—	—	—	—	—	—	—	—	—	—	—
Lomatium attenuatum (Sci. Name)	*Lomatium attenuatum*	—	—	—	—	—	—	—	—	—	—	—	—	P
Lomatium concinnum (Sci. Name)	*Lomatium concinnum*	—	P	—	—	—	—	—	—	—	—	—	—	—
Desert-parsley, Red-fruited	*Lomatium erythrocarpum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Desert-parsley, Greenman's	*Lomatium greenmanii*	—	—	—	—	—	—	—	—	C	—	—	—	—
Lomatium laevigatum (Sci. Name)	*Lomatium laevigatum*	—	—	—	—	—	—	—	—	P	—	—	P	—
Lomatium latilobum (Sci. Name)	*Lomatium latilobum*	—	P	—	—	—	—	—	—	—	—	P	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Lomatium nelsonianum (Sci. Name)	*Lomatium nelsonianum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lomatium oreganum (Sci. Name)	*Lomatium oreganum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lomatium, Peck's	*Lomatium peckianum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lomatium rollinsii (Sci. Name)	*Lomatium rollinsii*	—	—	P	—	—	—	—	—	P	—	—	P	—
Desert-parsley, Suksdorf's	*Lomatium suksdorfii*	—	—	—	—	—	—	—	—	P	—	—	P	—
Desert-parsley, Hoover's	*Lomatium tuberosum*	—	—	—	—	—	—	—	—	—	—	—	P	—
Luina serpentina (Sci. Name)	*Luina serpentina*	—	—	—	—	—	—	—	—	C	—	—	—	—
Lupinus aridus ssp. *ashlandensis* (Sci. Name)	*Lupinus aridus* ssp. *ashlandensis*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lupinus biddlei (Sci. Name)	*Lupinus biddlei*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lupinus crassus (Sci. Name)	*Lupinus crassus*	—	P	—	—	—	—	—	—	—	—	—	—	—
Lupinus cusickii (Sci. Name)	*Lupinus cusickii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Lygodesmia doloresensis (Sci. Name)	*Lygodesmia doloresensis*	—	P	—	—	—	—	—	—	—	—	—	—	—
Skeletonplant, Entrada	*Lygodesmia entrada*	—	P	—	—	—	—	—	—	—	—	P	—	—
Margaranthus lemmonii (Sci. Name)	*Margaranthus lemmonii*	P	—	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Blazing Star, Clay	*Mentzelia argillosa*	—	P	—	—	—	—	—	—	—	—	P	—	—
Mentzelia densa (Sci. Name)	*Mentzelia densa*	—	P	—	—	—	—	—	—	—	—	—	—	—
Stickleaf, Smooth	*Mentzelia mollis*	—	—	P	—	P	—	—	—	P	—	—	—	—
Stickleaf, Packard's	*Mentzelia packardiae*	—	—	—	—	—	—	—	—	P	—	—	—	—
Microseris howellii (Sci. Name)	*Microseris howellii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Mimulus gemmiparus (Sci. Name)	*Mimulus gemmiparus*	—	P	—	—	—	—	—	—	—	—	—	—	—
Monkey Flower, Stalk-leaved	*Mimulus patulus*	—	—	P	—	—	—	—	—	P	—	—	P	—
Monkeyflower, Pygmy	*Mimulus pygmaeus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Mirabilis rotundifolia (Sci. Name)	*Mirabilis rotundifolia*	—	P	—	—	—	—	—	—	—	—	—	—	—
Musineon lineare (Sci. Name)	*Musineon lineare*	—	—	—	—	—	—	—	—	—	—	P	—	—
Myosurus minimus ssp. *apus* (Sci. Name)	*Myosurus minimus* ssp. *apus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Naiad, Fish Lake	*Najas caespitosa*	—	—	—	—	—	—	—	—	—	—	P	—	—
Neolloydia erecto-centra var. *acunensis* (Sci. Name)	*Neolloydia erecto-centra* var. *acunensis*	C	—	—	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Neolloydia erecto-centra var. *erectocentra* (Sci. Name)	*Neolloydia erecto-centra* var. *erectocentra*	P	—	—	—	—	—	—	—	—	—	—	—	—
Neoparrya lithophila (Sci. Name)	*Neoparrya lithophila*	—	P	—	—	—	—	—	—	—	—	—	—	—
Notholaena lemmonii (Sci. Name)	*Notholaena lemmonii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Evening-primrose	*Oenothera acutissima*	—	P	—	—	—	—	—	—	—	—	P	—	—
Evening-primrose, Klein's	*Oenothera kleinii*	—	P	—	—	—	—	—	—	—	—	—	—	—
Oenothera organensis (Sci. Name)	*Oenothera organensis*	—	—	—	—	—	P	—	—	—	—	—	—	—
Evening-primrose, Wolf's	*Oenothera wolfii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Evening-primrose	*Oenothera psammophila*	—	—	P	—	—	—	—	—	—	—	—	—	—
Opuntia arenaria (Sci. Name)	*Opuntia arenaria*	—	—	—	—	—	P	—	—	—	—	—	—	—
Opuntia whipplei var. *multigeniculata* (Sci. Name)	*Opuntia whipplei* var. *multigeniculata*	P	—	—	—	P	—	—	—	—	—	—	—	—
Cholla, Wiggins	*Opuntia wigginsii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Oryctes nevadensis (Sci. Name)	*Oryctes nevadensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Oryzopsis swallenii (Sci. Name)	*Oryzopsis swallenii*	—	—	—	—	—	—	—	—	—	—	—	—	P
Parthenium tetraneuris (Sci. Name)	*Parthenium tetraneuris*	—	P	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Pectis imberbis (Sci. Name)	*Pectis imberbis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Pediocactus papyra-canthus (Sci. Name)	*Pediocactus papyra-canthus*	P	—	—	—	—	P	—	—	—	—	—	—	—
Pediocactus paradinei (Sci. Name)	*Pediocactus paradinei*	C	—	—	—	—	—	—	—	—	—	—	—	—
Pediocactus peeble-sianus var. fickeiseniae (Sci. Name)	*Pediocactus peeble-sianus var. fickeiseniae*	C												
Pediocactus winkleri (Sci. Name)	*Pediocactus winkleri*	—	—	—	—	—	—	—	—	—	—	C	—	—
Penstemon absorkensis (Sci. Name)	*Penstemon absorkensis*	—	—	—	—	—	—	—	—	—	—	—	—	P
Penstemon alamosensis (Sci. Name)	*Penstemon alamosensis*	—	—	—	—	—	P	—	—	—	—	—	—	—
Beardtongue	*Penstemon albifluvis*	—	C	—	—	—	—	—	—	—	—	C	—	—
Penstemon ammophilum (Sci. Name)	*Penstemon ammophilum*	—	—	—	—	—	—	—	—	—	—	P	—	—
Penstemon arenarius (Sci. Name)	*Penstemon arenarius*	—	—	—	—	P	—	—	—	—	—	—	—	—
Penstemon atwoodii (Sci. Name)	*Penstemon atwoodii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Penstemon barrettiae (Sci. Name)	*Penstemon barrettiae*	—	—	—	—	—	—	—	—	P	—	—	P	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Penstemon bicolor ssp. *bicolor* (Sci. Name)	*Penstemon bicolor* ssp. *bicolor*	—	—	—	—	P	—	—	—	—	—	—	—	—
Penstemon bicolor ssp. *roseus* (Sci. Name)	*Penstemon bicolor* ssp. *roseus*	P	—	—	—	P	—	—	—	—	—	—	—	—
Beardtongue, Red Canyon	*Penstemon bracteatus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beardtongue, Cache	*Penstemon compactus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beardtongue, Tunnel Springs	*Penstemon concinnus*	—	—	—	—	P	—	—	—	—	—	P	—	—
Beardtongue, Degener	*Penstemon degeneri*	—	P	—	—	—	—	—	—	—	—	—	—	—
Beardtongue	*Penstemon discolor*	C	—	—	—	—	—	—	—	—	—	—	—	—
Penstemon distans (Sci. Name)	*Penstemon distans*	P	—	—	—	—	—	—	—	—	—	—	—	—
Penstemon flowersii (Sci. Name)	*Penstemon flowersii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Penstemon, Amargosa	*Penstemon fruticiformis* ssp. *amargosae*	—	—	—	—	P	—	—	—	—	—	—	—	—
Penstemon gibbensii (Sci. Name)	*Penstemon gibbensii*	—	P	—	—	—	—	—	—	—	—	—	—	P
Beardtongue	*Penstemon glaucinus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Penstemon goodrichii (Sci. Name)	*Penstemon goodrichii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beardtongue, Graham	*Penstemon grahamii*	—	C	—	—	—	—	—	—	—	—	C	—	—
Beardtongue, Harrington	*Penstemon harringtonii*	—	C	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Beardtongue, Lemhi	*Penstemon lemhiensis*	—	—	P	P	—	—	—	—	—	—	—	—	—
Penstemon leptanthus (Sci. Name)	*Penstemon leptanthus*	—	—	—	—	—	—	—	—	—	—	P	—	—
Penstemon navajoa (Sci. Name)	*Penstemon navajoa*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beardtongue	*Penstemon pahutensis*	—	—	—	—	C	—	—	—	—	—	—	—	—
Penstemon parviflorus (Sci. Name)	*Penstemon parviflorus*	—	P	—	—	—	—	—	—	—	—	—	—	—
Penstemon peckii (Sci. Name)	*Penstemon peckii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Penstemon pudicus (Sci. Name)	*Penstemon pudicus*	—	—	—	—	P	—	—	—	—	—	—	—	—
Beardtongue	*Penstemon retrorsus*	—	C	—	—	—	—	—	—	—	—	—	—	—
Beardtongue, Wallowa	*Penstemon spatulatus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Beardtongue, Tidestrom	*Penstemon tidestromii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Beardtongue, Ward	*Penstemon wardii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Perideridia erythrorhiza (Sci. Name)	*Perideridia erythrorhiza*	—	—	—	—	—	—	—	—	P	—	—	—	—
Rock-daisy, Ajo	*Perityle ajoensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Perityle cernua (Sci. Name)	*Perityle cernua*	—	—	—	—	—	P	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Perityle cochisensis (Sci. Name)	*Perityle cochisensis*	P	—	—	—	—	—	—	—	—	—	—	—	—
Perityle saxicola (Sci. Name)	*Perityle saxicola*	P	—	—	—	—	—	—	—	—	—	—	—	—
Rockmat, Chelan	*Petrophytum cinerascens*	—	—	—	—	—	—	—	—	—	—	—	P	—
Phacelia argentea (Sci. Name)	*Phacelia argentea*	—	—	—	—	—	—	—	—	P	—	—	—	—
Phacelia, Beatley	*Phacelia beatleyae*	—	—	—	—	P	—	—	—	—	—	—	—	—
Phacelia	*Phacelia capitata*	—	—	—	—	—	—	—	—	P	—	—	—	—
Phacelia, Virgin	*Phacelia cephalotes*	P	—	—	—	—	—	—	—	—	—	P	—	—
Phacelia inconspicua (Sci. Name)	*Phacelia inconspicua*	—	—	P	—	P	—	—	—	—	—	—	—	—
Phacelia, Sticky	*Phacelia lenta*	—	—	—	—	—	—	—	—	—	—	—	P	—
Phacelia, Mono	*Phacelia monoensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Phacelia nevadensis (Sci. Name)	*Phacelia nevadensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Phacelia	*Phacelia submutica*	—	P	—	—	—	—	—	—	—	—	—	—	—
Phacelia verna (Sci. Name)	*Phacelia verna*	—	—	—	—	—	—	—	—	P	—	—	—	—
Phacelia	*Phacelia welshii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Phlox	*Phlox caryophylla*	—	P	—	—	—	P	—	—	—	—	—	—	—
Phlox, Clearwater	*Phlox idahonis*	—	—	C	—	—	—	—	—	—	—	—	—	—
Phlox sp. (Sci. Name)	*Phlox* sp.	—	—	—	—	—	—	—	—	—	—	—	—	P

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Pholisma	*Pholisma arenarium*	P	—	—	—	—	—	—	—	—	—	—	—	—
Pholisma sonorae (Sci. Name)	*Pholisma sonorae*	P	—	—	—	—	—	—	—	—	—	—	—	—
Physaria acutifolia var. purpurea (Sci. Name)	*Physaria acutifolia var. purpurea*	—	—	—	—	—	—	—	—	—	—	P	—	—
Physaria didymocarpa var. lyrata (Sci. Name)	*Physaria didymocarpa var. lyrata*	—	—	P	—	—	—	—	—	—	—	—	—	—
Physaria dornii (Sci. Name)	*Physaria dornii*	—	—	—	—	—	—	—	—	—	—	—	—	P
Plagiobothrys hirtus var. corallicarpus (Sci. Name)	*Plagiobothrys hirtus var. corallicarpus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Popcornflower	*Plagiobothrys hirtus var. hirtus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Popcornflower	*Plagiobothrys lamprocarpus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Semaphore Grass, Oregon	*Pleuropogon oregonus*	—	—	—	—	—	—	—	—	P	—	—	—	—
Blue Grass, Sea Cliff	*Poa unilateralis*	—	—	—	—	—	—	—	—	—	—	—	C	—
Jacob's Ladder	*Polemonium pauciflorum ssp. hinckleyi*	P	—	—	—	—	—	—	—	—	—	—	—	—
Polemonium pectinatum (Sci. Name)	*Polemonium pectinatum*	—	—	—	—	—	—	—	—	—	—	—	P	—
Combleaf	*Polyctenium williamsiae*	—	—	—	—	P	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Physaria bellii (Sci. Name)	*Physaria bellii*	—	P	—	—	—	—	—	—	—	—	—	—	—
Polygonum fusiforme (Sci. Name)	*Polygonum fusiforme*	P	—	—	—	—	—	—	—	—	—	—	—	—
Potentilla effusa var. *rupincola* (Sci. Name)	*Potentilla effusa* var. *rupincola*	—	P	—	—	—	—	—	—	—	—	—	—	—
Primula hunnewellii (Sci. Name)	*Primula hunnewellii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Primrose	*Primula nevadensis*	—	—	—	—	P	—	—	—	—	—	—	—	—
Primula wilcoxiana (Sci. Name)	*Primula wilcoxiana*	—	—	P	—	—	—	—	—	—	—	—	—	—
Proboscidea sabulosa (Sci. Name)	*Proboscidea sabulosa*	—	—	—	—	—	P	—	—	—	—	—	—	—
Scurf-pea	*Psoralea epipsila*	P	—	—	—	—	—	—	—	—	—	P	—	—
Psoralea pariensis (Sci. Name)	*Psoralea pariensis*	—	—	—	—	—	—	—	—	—	—	P	—	—
Psoralea trinervata (Sci. Name)	*Psoralea trinervata*	—	—	—	—	—	P	—	—	—	—	—	—	—
Psorothamnus polyadenius var. *jonesii* (Sci. Name)	*Psorothamnus polyadenius* var. *jonesii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Psorothamnus thompsonae var. *whitingii* (Sci. Name)	*Psorothamnus thompsonae* var. *whitingii*	—	P	—	—	—	—	—	—	—	—	—	P	——
Needlegrass, Porter's	*Ptilagrostis mongholica* ssp. *porteri*	—	P	—	—	—	—	—	—	—	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Alkali Grass, Parish's	Puccinellia parishii	P	—	—	—	—	P	—	—	—	—	—	—	—
Ranunculus austro-oreganus (Sci. Name)	Ranunculus austro-oreganus	—	—	—	—	—	—	—	—	P	—	—	—	—
Ranunculus reconditus (Sci. Name)	Ranunculus reconditus	—	—	—	—	—	—	—	—	P	—	—	P	—
Rorippa caycina (Sci. Name)	Rorippa calycina	—	—	—	P	—	—	P	—	—	—	—	—	P
Water Cress	Rorippa coloradensis	—	P	—	—	—	—	—	—	—	—	—	—	—
Rorippa columbiae (Sci. Name)	Rorippa columbiae	—	—	—	—	—	—	—	—	P	—	—	P	—
Rorippa subumbellata (Sci. Name)	Rorippa subumbellata	—	—	—	—	C	—	—	—	—	—	—	—	—
Rosa stellata (Sci. Name)	Rosa stellata	P	—	—	—	—	P	—	—	—	—	—	—	—
Rubus nigerrimus (Sci. Name)	Rubus nigerrimus	—	—	—	—	—	—	—	—	—	—	—	C	—
Rumex orthoneurus (Sci. Name)	Rumex orthoneurus	C	—	—	—	—	—	—	—	—	—	—	—	—
Salix arizonica (Sci. Name)	Salix arizonica	P	—	—	—	—	—	—	—	—	—	—	—	—
Saxifrage, Saddle Mountain	Saxifraga hitchcockiana	—	—	—	—	—	—	—	—	P	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Sclerocactus whipplei var. heilii (Sci. Name)	*Sclerocactus whipplei var. heilii*	—	—	—	—	—	P	—	—	—	—	—	—	—
Figwort	*Scrophularia coccinea*	—	—	—	—	—	C	—	—	—	—	—	—	—
Stonecrop	*Sedum moranii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Sedum oblanceolatum (Sci. Name)	*Sedum oblanceolatum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Selaginella utahensis (Sci. Name)	*Selaginella utahensis*	—	—	—	—	P	—	—	—	—	—	P	—	—
Schoencrambe barnebyi (Sci. Name)	*Schoencrambe barnebyi*	—	—	—	—	—	—	—	—	—	—	C	—	—
Groundsel, Intermediate	*Senecio dimorphophyllus var. intermedius*	—	P	—	—	—	—	—	—	—	—	P	—	—
Ragwort, Ertter's	*Senecio ertterae*	—	—	—	—	—	—	—	—	C	—	—	—	—
Senecio hesperius (Sci. Name)	*Senecio hesperius*	—	—	—	—	—	—	—	—	P	—	—	—	—
Groundsel, Huachuca	*Senecio huachucanus*	C	—	—	—	—	—	—	—	—	—	—	—	—
Senecio quaerens (Sci. Name)	*Senecio quaerens*	—	—	—	—	—	P	—	—	—	—	—	—	—
Shoshonea pulvinata (Sci. Name)	*Shoshonea pulvinata*	—	—	—	—	—	—	—	—	—	—	—	—	P
Sibara grisea (Sci. Name)	*Sibara grisea*	—	—	—	—	—	P	—	—	—	—	—	—	—
Checker-mallow, Meadow	*Sidalcea campestris*	—	—	—	—	—	—	—	—	P	—	—	—	—
Checker-mallow Nelson's	*Sidalcea nelsoniana*	—	—	—	—	—	—	—	—	P	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Sidalcea oregana var. *calva* (Sci. Name)	*Sidalcea oregana* var. *calva*	—	—	—	—	—	—	—	—	—	—	—	P	—
Silene clokeyi (Sci. Name)	*Silene clokeyi*	—	—	—	—	P	—	—	—	—	—	—	—	—
Catchfly, Cascade Head	*Silene douglasii* var. *oraria*	—	—	—	—	—	—	—	—	P	—	—	—	—
Catchfly, Red Canyon	*Silene petersonii* var. *minor*	—	—	—	—	—	—	—	—	—	—	C	—	—
Catchfly, Plateau	*Silene petersonii* var. *petersonii*	—	—	—	—	—	—	—	—	—	—	P	—	—
Silene rectiramea (Sci. Name)	*Silene rectiramea*	P	—	—	—	—	—	—	—	—	—	—	—	—
Silene regia (Sci. Name)	*Silene regia*	—	—	—	—	—	—	—	P	—	—	—	—	—
Silene scaposa var. *scaposa* (Sci. Name)	*Silene scaposa* var. *scaposa*	—	—	—	—	—	—	—	—	P	—	—	—	—
Silene seelyi (Sci. Name)	*Silene seelyi*	—	—	—	—	—	—	—	—	—	—	—	P	—
Silene spaldingii (Sci. Name)	*Silene spaldingii*	—	—	P	P	—	—	—	—	P	—	—	P	—
Sisyrinchium sarmentosum (Sci. Name)	*Sisyrinchium sarmentosum*	—	—	—	—	—	—	—	—	—	—	—	P	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Sophora gypsophila var. guadalupensis (Sci. Name)	*Sophora gypsophila var. guadalupensis*	—	—	—	—	—	P	—	—	—	—	—	—	—
Sophora leachiana (Sci. Name)	*Sophora leachiana*	—	—	—	—	—	—	—	—	P	—	—	—	—
Globe-mallow, Jones	*Sphaeralcea caespitosa*	—	—	—	—	P	—	—	—	—	—	P	—	—
Sphaeralcea procera (Sci. Name)	*Sphaeralcea procera*	—	—	—	—	—	P	—	—	—	—	—	—	—
Sphaeralcea psoraloides (Sci. Name)	*Sphaeralcea psoraloides*	—	—	—	—	—	—	—	—	—	—	P	—	—
Tansy	*Sphaeromeria compacta*	—	—	—	—	C	—	—	—	—	—	—	—	—
Tansy, Zion	*Sphaeromeria ruthiae*	—	—	—	—	—	—	—	—	—	—	P	—	—
Sagebrush, False Laramie	*Sphaeromeria simplex*	—	—	—	—	—	—	—	—	—	—	—	—	P
Spiranthes diluvialis (Sci. Name)	*Spiranthes diluvialis*	—	P	—	—	—	—	—	—	—	—	P	—	—
Streptanthus howellii (Sci. Name)	*Streptanthus howellii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Jewelflower, Lemmon's	*Streptanthus lemmonii*	P	—	—	—	—	—	—	—	—	—	—	—	—
Jewelflower	*Streptanthus squamiformis*	—	—	—	—	—	—	—	P	—	—	—	—	—
Stroganowia, Tiehm's	*Stroganowia tiehmii*	—	—	—	—	P	—	—	—	—	—	—	—	—
Sullivantia oregana (Sci. Name)	*Sullivantia oregana*	—	—	—	—	—	—	—	—	P	—	—	P	—
Synthyris missurica ssp. *hirsuta* (Sci. Name)	*Synthyris missurica* ssp. *hirsuta*	—	—	—	—	—	—	—	—	P	—	—	—	—

Table H-1. Special Status Species List (continued)

Part B: Candidate and Proposed Candidate Species

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Kittentails	*Synthyris ranunculina*	—	—	—	—	C	—	—	—	—	—	—	—	—
Talinum marginatum (Sci. Name)	*Talinum marginatum*	P	—	—	—	—	—	—	—	—	—	—	—	—
Talinum validulum (Sci. Name)	*Talinum validulum*	P	—	—	—	—	—	—	—	—	—	P	—	—
Tauschia hooveri (Sci. Name)	*Tauschia hooveri*	—	—	—	—	—	—	—	—	—	—	—	P	—
Tauschia, Howell's	*Tauschia howellii*	—	—	—	—	—	—	—	—	P	—	—	—	—
Thalictrum heliophilum (Sci. Name)	*Thalictrum heliophilum*	—	P	—	—	—	—	—	—	—	—	—	—	—
Thelesperma pubescens (Sci. Name)	*Thelesperma pubescens*	—	—	—	—	—	—	—	—	—	—	—	—	P
Thelesperma subnudum var. *alpinum* (Sci. Name)	*Thelesperma subnudum* var. *alpinum*	—	—	—	—	—	—	—	—	—	—	P	—	—
Thelypody, Clay	*Thelypodiopsis argillacea*	—	—	—	—	—	—	—	—	—	—	P	—	—
Thelypodium eucosmum (Sci. Name)	*Thelypodium eucosmum*	—	—	—	—	—	—	—	—	P	—	—	—	—
Thelypodium howellii var. *spectabilis* (Sci. Name)	*Thelypodium howellii* var. *spectabilis*	—	—	—	—	—	—	—	—	C	—	—	—	—
Thelypody, Jaeger's (wavy-leaf)	*Thelypodium repandum*	—	—	C	—	—	—	—	—	—	—	—	—	—

Common Name	Scientific Name	AZ	CO	ID	MT	NV	NM	ND	OK	OR	SD	UT	WA	WY
Thlaspi montanum var. siskiyouense (Sci. Name)	Thlaspi montanum var. siskiyouense	—	—	—	—	—	—	—	—	P	—	—	—	—
Townsendia jonesii var. tumulosa (Sci. Name)	Townsendia jonesii var. tumulosa	—	—	—	—	P	—	—	—	—	—	—	—	—
Townsendia sp. (Sci. Name)	Townsendia sp. (Nye Co.)	—	—	—	—	P	—	—	—	—	—	—	—	—
Townsendia sp. (Sci. Name)	Townsendia sp.	—	—	—	—	—	—	—	—	—	—	—	—	P
Tradescantia ozarkana (Sci. Name)	Tradescantia ozarkana	—	—	—	—	—	—	—	P	—	—	—	—	—
Trifolium andersonii var. friscanum (Sci. Name)	Trifolium andersonii var. friscanum	—	—	—	—	—	—	—	—	—	—	C	—	—
Trifolium barnebyi (Sci. Name)	Trifolium barnebyi	—	—	—	—	—	—	—	—	—	—	—	—	P
Trifolium leibergii (Sci. Name)	Trifolium leibergii	—	—	—	—	—	—	—	—	P	—	—	—	—
Clover, Owyhee	Trifolium owyheense	—	—	P	—	—	—	—	—	P	—	—	—	—
Clover, Thompson	Trifolium thompsonii	—	—	—	—	—	—	—	—	—	—	—	P	—
Trisetum orthochaetum (Sci. Name)	Trisetum orthochaetum	—	—	—	P	—	—	—	—	—	—	—	—	—
Vauquelinia pauciflora (Sci. Name)	Vauquelinia pauciflora	P	—	—	—	—	P	—	—	—	—	—	—	—
Xylorhiza cronquistii (Sci. Name)	Xylorhiza cronquistii	—	—	—	—	—	—	—	—	—	—	P	—	—

Appendix I
Target Plant Species

Common Name	Scientific Name	State(s)
Grasses		
Bermudagrass	*Cynodon dactylon*	Arizona
Bluegrass, Annual	*Poa annua*	South Dakota
Cheatgrass	*Bromus tectorum*	Idaho
Foxtail, Giant	*Setaria faberi*	South Dakota
Johnsongrass	*Sorghum halepense*	Arizona, Nevada
Medusahead	*Taeniatherum asperum*	Idaho, Utah
Oats, Wild	*Avena fatua*	South Dakota
Quackgrass	*Agropyron repens*	South Dakota
Forbs		
Amaranth, Palmer	*Amaranthus palmeri*	New Mexico
Bindweed, Field	*Convolvulus arvensis*	Nevada, North Dakota, South Dakota
Bindweed, Hedge	*Convolvulus sepium*	South Dakota
Buffalobur	*Solanum rostratum*	Nevada
Burdock	*Arctium minus*	Colorado
Camphorweed	*Heterotheca subaxillaris*	Arizona
Cocklebur	*Xanthium pennsylvanicum*	Nevada, North Dakota, South Dakota, Utah, Wyoming
Cocklebur, Mexican	*Xanthium* sp.	Arizona
Cress, Hoary (Whitetop)	*Cardaria draba*	Nevada, Wyoming
Deathcamas	*Zigadenus gramineus*	Idaho, Montana, Wyoming
Dodder	*Cuscuta* spp.	South Dakota
Fieldcress, Austrian	*Rorippa austriaca*	Nevada
Gumweed, Curly Cup	*Grindelia squarrosa*	Utah, Wyoming
Halogeton	*Halogeton glomeratus*	Nevada, Wyoming
Hemlock, Poison	*Conium maculatum*	Nevada
Hemlock, Water	*Cicuta* sp.	Nevada
Hemp	*Cannabis sativa*	North Dakota
Henbane, Black	*Hyoscyamus niger*	Colorado, Utah
Hounds Tongue	*Cynoglossum officinale*	Colorado, Utah
Iris	*Iris* spp.	Nevada, Wyoming
Ivy, Poison	*Toxicodendron radicans*	Colorado
Knapweed	*Centaurea* spp.	Colorado, Nevada
Knapweed, Diffuse	*Centaurea diffusa*	Idaho, Nevada, Wyoming
Knapweed, Russian	*Centaurea repens*	Nevada, North Dakota, South Dakota, Utah, Wyoming
Knapweed, Spotted (or Squarose)	*Centaurea maculosa*	North Dakota, South Dakota, Utah, Wyoming
Kochia	*Kochia scoparia*	Arizona, Wyoming
Larkspur, Desert		Arizona, New Mexico
Licorice	*Glycyrrhiza lepidota*	Nevada
Mallow, Little	*Malva parviflora*	Arizona
Milkweed, Poison	*Asclepius* spp.	Utah
Mustard, Tumble	*Sisymbrium altissimum*	Wyoming
Mustard, Wild	*Brassica kaber, B. nigra, and B. juncea*	South Dakota
Nettle, Horse	*Solanum carolinese and S. elaeagnifolium*	Nevada, South Dakota
Nightshade	*Solanum* spp.	New Mexico
Pennycress, Field	*Thlaspi arvense*	South Dakota
Pepperweed, Perennial	*Lepidium latifolium*	Wyoming

Common Name	Scientific Name	State(s)
Ragweed, Common	*Ambrosia artemisiifolia*	Idaho, Wyoming
Ragwort, Tansy	*Senecio jacobaea*	Idaho, Oregon, Wyoming
Sowthistle, Perennial	*Sonchus arvensis*	North Dakota, South Dakota
Spurge, Leafy	*Euphorbia esula*	Colorado, Nevada, North Dakota, South Dakota, Utah
Starthistle, Iberian	*Centaurea iberica*	Nevada
Starthistle, Purple	*Centaurea calcitrapa*	Nevada
Starthistle, Yellow	*Centaurea solstitialis*	Nevada
Sunflower	*Helianthus annuus*	Arizona
Thistle, Canada	*Circium arvense*	Colorado, Nevada, North Dakota, South Dakota, Utah
Thistle, Musk	*Carduus nutans*	Nevada, North Dakota, South Dakota, Utah, Wyoming
Thistle, Russian	*Salsola kali*	Arizona, Nevada, New Mexico
Thistle, Scotch	*Onopordum acanthium*	Nevada, Utah
Thistle, Blue	*Echium vulgare*	Colorado
Thistle, Plumeless	*Carduus acanthoides*	South Dakota
Toadflax	*Linaria* spp.	Colorado, Nevada
Vine, Puncture	*Tribulus terrestris*	Nevada
Woad, Dyers	*Isatis tinctoria*	Colorado
Shrubs and Trees		
Acacia	*Acacia* spp.	Arizona
Alder	*Alnus* spp.	Idaho
Berry, Thimble	*Rubus parviflorus*	Idaho
Broom, Desert	*Baccharis sarothroides*	Arizona
Burroweed	*Haplopappus tenuisectus*	Arizona
Camelthorn	*Alhagi camelorum*	Arizona, Nevada
Ceanothus	*Ceanothus* spp.	Arizona
Cholla	*Opuntia* spp.	New Mexico
Creosotebush	*Larrea tridentata*	Arizona, New Mexico
Elderberry	*Sambucus canadensis*	Idaho
Greasewood	*Sarcobatus vermiculatus*	Nevada, Oregon, Utah
Juniper	*Juniperus* spp.	Arizona, Idaho, Nevada, Oregon, Utah
Juniper, One-seed	*Juniperus monosperma*	Nevada, New Mexico
Locust, New Mexican	*Robinia neomexicana*	Arizona
Manzanita	*Arctostaphylos* spp.	Oregon
Maple	*Acer* spp.	Idaho
Mesquite	*Prosopis juliflora and P. velutina*	Arizona, New Mexico
Mulberry	*Morus* spp.	Colorado
Nine-bark	*Physocarpus malvaceus*	Idaho
Oak, Gambel	*Quercus gambelii*	Colorado
Oak, Shinnery	*Quercus havardii*	New Mexico
Oak, Shrub Live	*Quercus turbinella*	Arizona, New Mexico
Peaweed, Austrian	*Sphaerophysa salsula*	Nevada
Pine, Pinyon	*Pinus edulis*	Nevada
Rabbitbrush	*Chrysothamnus* spp.	Colorado, Idaho, Nevada, New Mexico, Oregon
Rose	*Rosa* spp.	Idaho
Sage, Mediterranean	*Salvia aethiopis*	Nevada
Sagebrush	*Artemisia* spp.	Arizona, Colorado, Nevada, Oregon, Wyoming
Sagebrush, Sand	*Artemisia filifolia*	Idaho, New Mexico
Sagebrush, Big	*Artemisia tridentata*	Idaho, Nevada, New Mexico, Utah
Saltbush, Fourwing	*Atriplex canescens*	Arizona

Common Name	Scientific Name	State(s)
Saltcedar (tamarisk)	*Tamarix pentandra*	Arizona, Colorado, Nevada, New Mexico
Snakeweed	*Gutierrezia sarothrae*	Arizona, New Mexico, Utah, Colorado
Spray, Ocean	*Holodiscus discolor*	Idaho
Tarbush	*Flourensia cernua*	New Mexico
Weed, Klamath	*Hypericum perforatum*	Nevada
Wormwood, Absinth	*Artemisia absinthim*	North Dakota
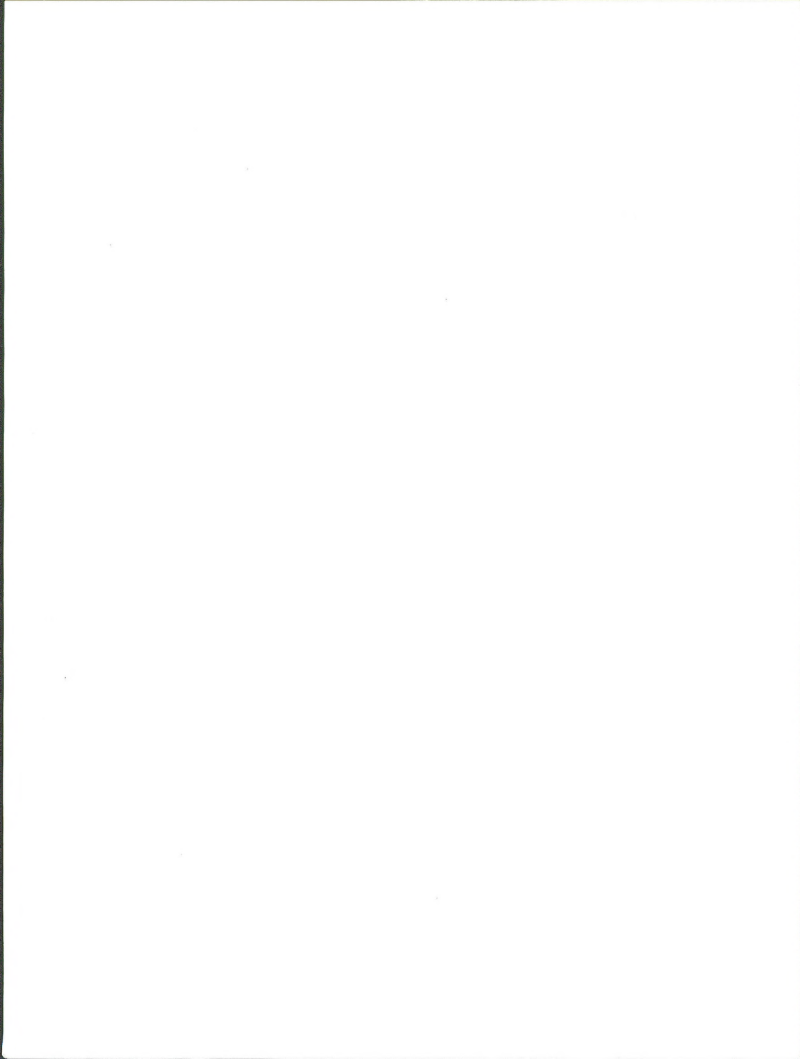
Appendix J
References for General and Specific Program Direction Concerning Use of Renewable Resource Improvements

General and specific program direction (policy, required procedures, and standards) concerning use of renewable resource improvements is contained in several Manual Sections and Handbooks. The following list of references provides a general index to information for Bureau managerial and staff personnel to use. Additional specific information is contained in the material referenced below:

(1) Handbook H-1740-1—Renewable Resource Improvement and Treatment Guidelines and Procedures.

(2) Manual Section 1741—Renewable Resource Improvements, Practices, and Standards.

(3) Handbook H-1741-1—Fencing.

(4) Manual Section 1742—Emergency Fire Rehabilitation.

(5) Handbook H-1742-1—Emergency Fire Rehabilitation.

(6) Manual Section 1743—Renewable Resource Investment Analysis System.

(7) Handbook H-1743-1—Resource Investment Analysis: User Handbook for the SageRam Computer Program.

(8) Manual Section 1112—Safety.

(9) Manual Section 1510—Procurement.

(10) Manual Section 1601—Bureau Planning System.

(11) Manual Section 1617—Resource Management Plan Approval, Use, and Modification.

(12) Manual Section 1619—Activity Plan Coordination.

(13) Manual Section 1620-1625—Supplemental Program Guidance.

(14) Manual Section 2920—Leases, Permits, and Easements.

(15) Manual Section 4100—Grazing Administration.

(16) Manual Section 4120—Grazing Management.

(17) Handbook H-4120-1—Grazing Management.

(18) Manual Section 5000—Forest Management.

(19) Manual Section 5400—Sales of Forest Products.

(20) Manual Section 6500—Wildlife Management.

(21) Manual Section 6780—Habitat Management Plans.

(22) Manual Section 6840—Threatened and Endangered Wildlife.

(23) Manual Section 7000—Soil, Water, and Air Management.

(24) Manual Section 9100—Engineering.

(25)   Manual Section 9101—Facility Planning.

(26)   Manual Section 9102—Facility Design.

(27)   Manual Section 9103—Facility Construction.

(28)   Manual Section 9104—Facility Maintenance.

(29)   Manual Section 9114—Trails.

(30)   Manual Section 9132—Operational Signs.

(31)   Manual Section 9172—Water Control Structures.

(32)   Handbook H-9172-1—Water Control Structures—Guidelines for Design.

(33)   Handbook H-9172-2—Water Control Structures—Guidelines for Construction.

(34)   Manual Section 9177—Maintenance and Safety of Dams.

(35)   Handbook H-9177-1—Performing Condition Surveys for Earth Embankment Dams.

(36)   Handbook H-9177-2—Preparing Emergency Action Plans.

(37)   Handbook H-9177-3—Reporting Dam Failures.

(38)   Manual Section 9182—Wastewater Treatment.

(39)   Manual Section 9183—Municipal/Community Related Solid Waste.

(40)   Manual Section 9184—Drinking Water Supply.

(41)   Manual Section 9220—Integrated Pest Management.

(42)   Manual Section 9222—Chemical Pest Control.

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